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TUMORS OF THE POSTERIOR PORTION OF THE THIRD VENTRICLE

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NEOPLASMS located within the posterior portion of the 3rd ventricle include those that originate from its posterior wall and from the pineal body. This paper is a review of the clinical, pathological, anatomical and therapeutic aspects of these lesions in 32 patients as they were studied at the University Hospital from the years 1930 to 1951. Nineteen of these tumors were verified histologically and 13 were diagnosed by clinical and ventriculographic findings. Tumors of the thalamus, the splenium of the corpus callosum, the choroid plexus of the 3rd ventricle and the anterior portion of the 3rd ventricle are not considered.

CLINICAL CONSIDERATIONS

The series is comprised of 24 males and 8 females whose ages varied from 14 months to 64 years. Although the average age was 21, 18 of these patients were under 16 years of age. The longest preoperative duration of symptoms was 5 years and was characterized by progressive loss of vision; the shortest duration was 10 days, introduced acutely by headache, nausea and vomiting. Prior to treatment the average duration of illness was 11 months. The predominating symptoms were precipitated by obstruction of the aqueduct of Sylvius (Fig. 1) resulting in either acute or chronic increased intracranial pressure which was manifested by headache, nausea, vomiting, papilledema and blurring of vision. In some chronic cases secondary optic atrophy developed.

Alterations in ocular movements were observed in 15 patients. Limitation to complete absence of upward automatic conjugate gaze was noted in 7 of the pathologically verified cases and in 8 cases diagnosed by clinical and ventriculographic findings. Among these 15 patients there was loss of downward conjugate gaze in 1 case of teratoma and pinealoma (Fig. 2), and in 1 case diagnosed clinically. Mydriasis and inequality of the pupils were equally frequent, each being noted in 9 patients. Diminished or absent pupillary light reflex was noted unilaterally or bilaterally in 20 cases; but in only 14 could alteration of the accommodation responses be shown (Table 1). One patient showed typical Argyll-Robertson pupils while 2 others had Argyll-Robertson-like pupillary responses, i.e., poor reaction to light yet good in accommodation.

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TABLE 1

Summary of the eye signs

Cases	Pupils			Diplopia	Nystagmus	Limitation of Upward Gaze
	Dilated	Impaired Reaction				
		Light	Accommodation			
Posner & Horrax[19] 16 cases	31% 5	50% 8	12% 2	25% 4	37.5% 6	31% 5—definite 1—?
Rand & Lemmen						
Pathologically verified 19 cases	21% 4	68% 13	42% 8	37% 7	32% 6	37% 7
Not pathologically verified 13 cases	38% 5	54% 7	46% 6	54% 7	23% 3	62% 8
Total 32 cases	28% 9	63% 20	44% 14	44% 14	28% 9	47% 15

	Sixth Nerve Palsy	Papilledema	Visual Field Changes (including Blind Spots)	Secondary Optic Atrophy
Posner & Horrax[19] 16 cases	25% 4	56% 9 1?	19% 3	
Rand & Lemmen				
Pathologically verified 19 cases	47% 9	74% 14	53% 10	21% 4
Not pathologically verified 13 cases	38% 5	77% 10	38% 5	0
Total 32 cases	44% 14	75% 24	47% 15	13% 4

Cerebellar symptoms such as asynergia, dysmetria, ataxia, hypotonia and disturbances of balance were evident in 11 cases. Nystagmus or nystagmoid movements appeared in 6 patients with histologically verified neoplasms and in 3 of those diagnosed by clinical examination.

Endocrine and hypothalamic disturbances were found in 17 cases: 1 female suffered from amenorrhea and another had poorly developed secondary sexual characteristics; 2 males had infantile genitalia with hypoplasia of the testes. The syndrome of precocious puberty was not diagnosed in any of the 32 cases. Diabetes insipidus was evident in 3 cases and hypersomnia in 10.

Extrapyramidal and pyramidal signs were observed in 13 cases. In 3 patients there was evidence for spinal cord involvement with a sensory level and motor changes. Hearing was impaired unilaterally or bilaterally in 5 cases.

ANATOMICAL BASIS FOR CLINICAL FINDINGS

Crosby and Henderson,[5] in their study of ocular movements in the Macaca mulatta, found that areas 18 and 19 are anatomically and functionally related to the ocular centers of the midbrain through the internal corticotectal tracts. The fibers concerned with upward conjugate deviation of the eyes terminate in the rostromedial portion of the superior colliculus and those mediating downward conjugate deviation terminate in its caudolateral portion. Tumors in the pineal region may cause loss of automatic upward deviation of the eyes (Parinaud's syndrome), by pressure upon or invasion of the rostral portions of the superior colliculi or interruption of fibers of the internal corticotectal tracts which terminate in the same region (Fig. 1). Pressure upon or invasion of the caudolateral portion of the superior colliculi will produce loss of downward conjugate deviation (Fig. 2). Automatic horizontal conjugate deviation of the eyes mediated by the corticotegmental tracts is rarely lost because these fibers pass through the tegmentum of the midbrain.[4,14]

Involvement of the oculomotor nucleus may result in paralysis or paresis of the individual muscles supplied by this nerve. Since the tecto-oculomotor fibers from the anterior part of the superior colliculus terminate in the anterior portion of the nucleus of the 3rd nerve, and the fibers from the posterior part of the superior colliculus terminate in the caudal portions of the oculomotor nucleus, involvement of this tract, or its termination in this nucleus, will also result in interference with automatic eye movements either upward or downward, depending upon the portion involved. Voluntary vertical movement of the eyes may be lost if there is interference with the corticobulbar tracts which terminate in the nucleus of the 3rd nerve. Absence of reflex conjugate movements of the eyes may be explained by involvement of the fibers of the medial longitudinal fasciculus which end at this level in the oculomotor nucleus.[4]

Pupillary reflex changes, including those of the Argyll-Robertson type, are also explainable on an anatomical basis. The earliest changes result from involvement of the afferent fibers from the retina which pass through the pretectal region to the Edinger-Westphal nucleus. This nucleus is divided into a rostral and a caudal part. The former regulates the reflex to light and the latter controls the iris and ciliary muscles in accommodation. The cells of these nuclei are of the "preganglionic type" and their fibers course to the eyes in association with those of the oculomotor nerve.[6] The Argyll-Robertson phenomenon, therefore, results from interference with the pretectal region and the rostral portions of the Edinger-Westphal nucleus.

Cerebellar signs and symptoms are considered to be the direct result of pressure upon, or invasion of, the cerebellar hemispheres or the superior cerebellar peduncles by the neoplasm (Fig. 2). Nystagmus is not generally

FIG. 1. (A) Sagittal section of brain showing tumor (teratoma) bed after surgical removal. The tumor had compressed the anterior half of the superior colliculus (arrow) and posterior portion of the hypothalamus and thalamus. The aqueduct of Sylvius was occluded. (B) Photograph of the teratoma removed at operation.

thought to be part of the superior cerebellar peduncle syndrome,[26] and is believed to represent a basic incoordination of eye movements similar to incoordinated movements of skeletal musculature.

According to Fulton and others,[9] destruction of the supraoptic nuclei of the anterior hypothalamus or its connections with the posterior lobe of the pituitary gland results in diabetes insipidus. Hypersomnia in these cases is associated with involvement of the posterior portion of the hypothalamus and/or the dorsal and ventral areas of the anterior region of the midbrain (see Cases 2 and 3).

Interference of the corticospinal motor system provokes hemiparesis and extrapyramidal findings. Auditory symptoms develop with dysfunction of the inferior colliculi and their immediately associated pathways. Lesions of the spinal cord or cauda equina with severe radicular pain are uncommon but may be caused by "seeding" of the neoplasm in the spinal subarachnoid

FIG. 2. Transverse section of midbrain showing hemorrhage into tumor bed. The patient had loss of both upward and downward conjugate gaze. (Arrow points to tectum of midbrain.)

space.[1] Palsy of the abducens nerve as seen in these patients can be accounted for by pressure upon its fibers by the lateral branches of the basilar artery in the presence of increased intracranial pressure.[7]

ROENTGENOLOGICAL FINDINGS

In 60 per cent of these cases the plain roentgenograms of the skull demonstrated changes incident to increased intracranial pressure, i.e., erosion of the dorsum sellae, separation of one or more sutures and increased convolutional markings of the inner table of the skull. Solid calcification of the pineal body proper was found in 7 patients, whose ages varied from 13 to 15 years. Of these calcified lesions, 3 were histologically confirmed as pinealomas (Fig. 7) and 1 was a pinealoblastoma; the remaining 3 were diagnosed clinically. Abnormal calcification appearing as scattered foci in the

region of the pineal body was seen in 5 cases; 2 of these were confirmed histologically as pinealoblastoma and 1 as pinealoma.

Ventriculograms performed in 31 cases revealed moderate to extreme dilatation of the lateral ventricles. The original ventriculograms were available to the authors for study in 15 of the cases of pathologically verified tumors and in 12 of those diagnosed clinically. In the former group there was partial obstruction of the foramina of Monro in 2 instances and in all 15 a complete obstruction of the aqueduct of Sylvius. In 4 cases the neoplasm filled the posterior quarter, in 6 approximately the posterior half and in 5 about the posterior three-quarters of the 3rd ventricle. The visible outline of the tumors appeared smooth and regular in 13 cases and irregular in 2. The anterior portion of the 3rd ventricle showed various degrees of suprasellar herniation. Encroachment upon the medial inferior walls of the lateral ventricles was noted in 6 cases, 5 of which had the largest filling defects in the posterior portion of the 3rd ventricle. A normal suprapineal recess was present in 1 case of teratoma, a pseudosuprapineal recess in 3 cases and in 11 cases the recess was obliterated.

Those tumors diagnosed clinically also showed complete obstruction of the aqueduct in all cases. The foramina of Monro were partially obstructed in 1 case. In 7 cases the neoplasm occupied the posterior one-quarter, in 3 approximately the posterior half and in 2 the posterior three-quarters of the 3rd ventricle. The visible outline of the tumor was smooth and regular in 9 instances and irregular in 3. Encroachment upon the medial inferior wall of the lateral ventricles was found in 1 case. A normal suprapineal recess was present in 2, a pseudosuprapineal recess in 5 and complete obliteration in 5.

PATHOLOGY

Histologically the 19 tumors were classified as: 9 pinealomas, 5 pinealoblastomas, 4 teratomas and 1 undifferentiated glioma. All were thought to have arisen from the pineal body or from midline structures of the posterior wall of the 3rd ventricle with involvement of the surrounding structures being secondary. The pineal body, having been replaced by neoplasm, could not be identified in 14 cases at postmortem examination; it was present in 1 case of pinealoma in which the tumor arose from the posterior wall of the 3rd ventricle beneath the aqueduct of Sylvius.

The pinealomas in this series conformed to histologic type. There were small and large cells surrounded by connective-tissue network. The small cells had round nuclei with dense chromatin network and scanty cytoplasm and resembled lymphocytes. The large epithelioid cells had distinct cytoplasm, their nuclei were poor in chromatin and contained one or two nucleoli. The large cells were grouped in a definite mosaic pattern (Fig. 8). The pinealoblastomas, on the other hand, were composed of small cells with round nuclei containing a moderate amount of chromatin and had no typical arrangement. Mitotic figures were common and there was little connective-tissue stroma. The 4 teratomas were complex tumors with derivatives of the

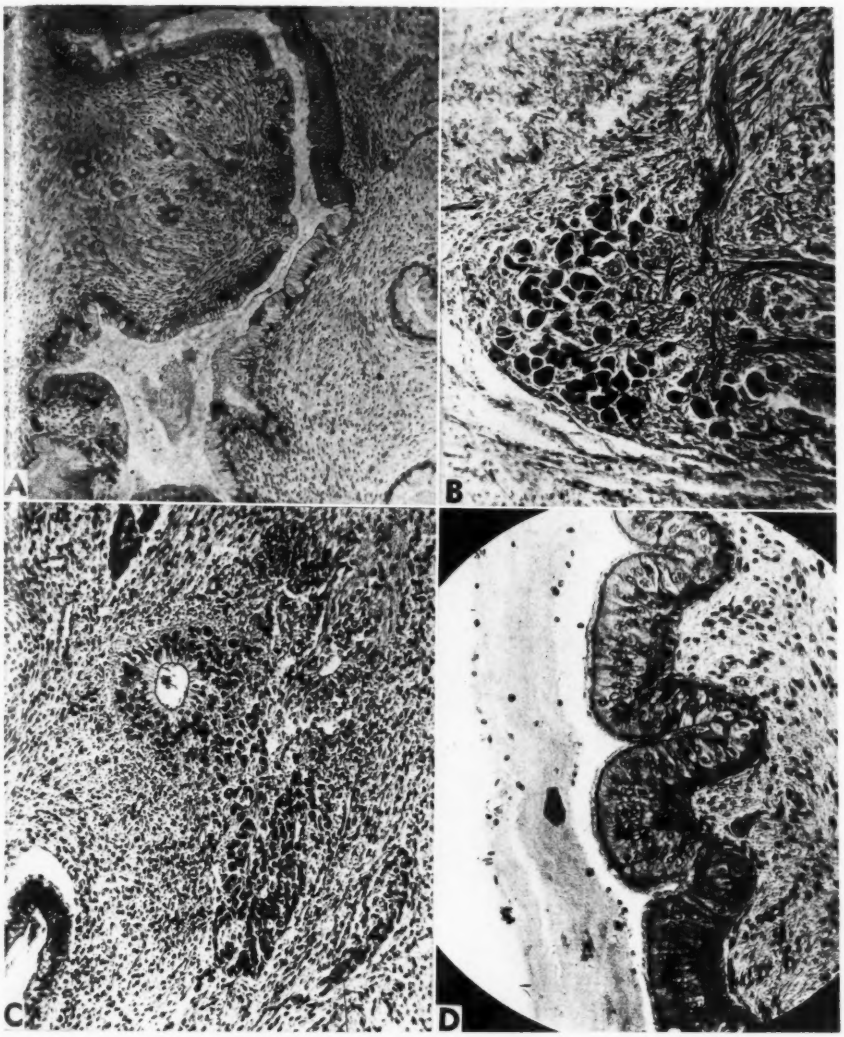

FIG. 3. Photomicrographs of representative sections of teratoma of pineal body. (A) Cyst lined by stratified squamous and columnar epithelium. (B) Section of ganglion cells and nerve fibers. (C) Section of gland formation. (D) Section of ciliated pseudostratified columnar epithelium.

three germ layers (Fig. 3). They contained a variety of structures such as brain tissue with ganglia and glial tissue, nerve fibers, retina, hair follicles, muscle fibers, cartilage, bone, fat, lymphoid, connective and vascular tissue, stratified squamous and columnar epithelium—the latter resembling that of the respiratory and intestinal tracts.

The histological patterns of calcification in these various tumors have been considered in greater detail elsewhere.[17] The most frequent type was that found in the walls of blood vessels.

CASE REPORTS

Case 1. C.M., a 14-year-old white male, was admitted to University Hospital on April 14, 1945. In November, 1944 progressive weakness and fatigue had developed, followed in 4 months by severe headache, double vision, increased thirst, polyuria, intermittent vomiting, and loss of 20 lbs. in weight.

Examination. He was a small, emaciated boy who was 4 ft. 6 in. tall and weighed 57 lbs. The genitalia were small and poorly developed and there was complete absence of secondary sexual characteristics. Mentally he was confused and lethargic. The pupils were round, equal and reacted to light; however, extraocular movements could not be examined satisfactorily. A 6th nerve palsy was present on the right. There was bilateral papilledema. The temporal fields were contracted bilaterally. The deep reflexes were hyperactive and Babinski's sign was present bilaterally.

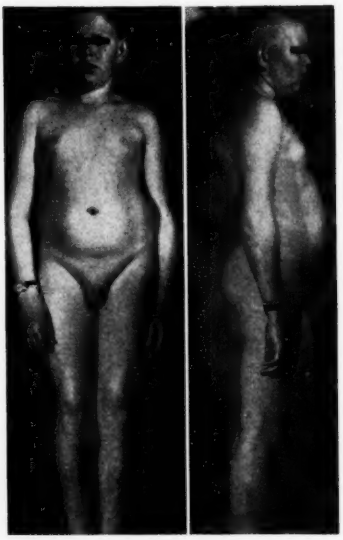

FIG. 4. *Case 1.* Photograph of patient at the age of 20, showing adiposogenital dystrophy.

Specific gravity of urine was 1.003; otherwise it was normal. BMR was minus 33 per cent. Total protein content of the ventricular fluid was 14 mg. per cent. Reaction to Kahn test was negative.

Roentgenograms of the skull showed scattered areas of calcification in the midline in the region of the pineal body. Minimal osteoporosis of the dorsum sellae was noted. A ventriculogram demonstrated a space-occupying lesion in the midline, which obliterated the 3rd ventricle with the exception of its superior anterior portion. The lateral ventricles were dilated symmetrically.

Operation. On April 20, 1945, a right lateral ventriculocisternostomy was performed (Torkildsen procedure).

Postoperative course was uneventful. The patient was given a total of 3000 r in air to each right and left skull field (200 Kv, 0.5 mm. Cu+1 mm. Al filter, F.S.D. 50, H.V.L. 0.9 mm., 42–52 r/min., field size 11 by 12 cm. over a 30-day period). He improved rapidly and was discharged June 6, 1945.

Five years later, at the age of 20, the patient was re-examined. There were no symptoms of increased intracranial pressure, but moderate polydipsia and polyuria continued and he had increased sensitivity to cold. He had grown only $4\frac{3}{4}$ in. (height 4 ft., $10\frac{3}{4}$ in.) but had gained 48 lbs. (105 lbs.). The scalp hair was coarse and dry and the eyebrows were sparse. Axillary, pubic and body hair were absent. Epilation had followed roentgen therapy. The genitalia were of an infantile type (Fig. 4).

Mentally he was dull and childish. The pupils were large and reacted sluggishly to light and in accommodation. There was limitation of upward automatic conjugate

deviation of the eyes, but voluntary movements were normal. Nystagmoid jerks were present on vertical gaze. There was pallor of the temporal margin of the right optic disc.

BMR was minus 27 per cent, fasting blood sugar 48 mg. per cent, total serum cholesterol 288 mg. per cent, and Hb. 77 per cent. Before injection of 0.3 cc. of 1/1000 adrenalin, the eosinophilic count was 311/c.mm.; 4 hrs. later it dropped very slightly to 211. The 17 ketosteroids were 316 mg./24 hrs. Reaction to Kahn test was negative.

Roentgenograms of the skull showed the radio-opaque tube placed at the time of the right lateral ventriculocisternostomy. The volume and character of the calcification in the pineal region and the shape of the dorsum sellae remained unchanged. Wrist and knee roentgenograms showed osseous maturation corresponding to the age of 14 years.

Comment. This patient is presented as an example of adiposogenital dystrophy (Fröhlich's syndrome) apparently caused by a large tumor in the posterior three-quarters of the 3rd ventricle. The clinical and laboratory findings are compatible with severe panhypopituitarism and have remained essentially unchanged since the Torkildsen operation and irradiation therapy. He represents the only patient in this series with severe endocrine dysfunction.

Case 2. N.P., a 23-year-old white female was admitted on Aug. 17, 1939. Five years previously she had been treated unsuccessfully with hormones (type unknown) for primary amenorrhea. Two years before admission she became blind in her left eye and during the last 6 months failing vision had developed on the right, accompanied by progressive hypersomnia, periods of polydipsia and generalized headaches.

Examination. She was undersized, with childlike facial expressions. Axillary hair was lacking and the pubic hair was sparse, but the breasts were developed normally. She was cooperative and intelligent. The pupils were about equal; reaction to direct light was absent on the left, but normal on the right. The accommodation-convergence reaction was prompt bilaterally. There was primary optic atrophy in the left eye and beginning atrophy in the right. The left eye was amaurotic and the right visual field was contracted centrally, suggesting early temporal hemianopsia. The skeletal musculature was moderately hypotonic, but motor power and coordination were normal. Reflexes were hypoactive and there was a bilateral Babinski's sign. Sensory modalities were intact.

Roentgenograms of the skull revealed erosion and enlargement of the left optic foramen (Fig. 5). Pneumoencephalograms and ventriculograms suggested a suprasellar lesion; however, the 3rd and 4th ventricles were visualized incompletely.

Specific gravity was not noted, but otherwise urine was normal. Hb. 80 per cent; fasting blood sugar 64 mg./100 cc.; blood Kahn reaction negative. BMR minus 23 per cent. CSF pressure during lumbar puncture, 90 mm. of water; the fluid contained 30 lymphocytes/c.mm.; total protein content 137 mg. per cent; colloidal gold curve 5555533210; mastic curve 444443; globulin 4+; Kahn reaction negative.

Operation. A right frontal osteoplastic craniotomy was performed under general anesthesia on Aug. 19, 1937. The left optic nerve and chiasma were diffusely infiltrated with a grayish granular neoplasm. The right optic nerve was not involved directly, but appeared compressed by the tumor on the left. Approximately 15 gm. of the lesion were removed in an attempt to decompress the right optic nerve.

Course. The patient died on the 1st postoperative day.

Autopsy. The brain weighed 1390 gm. A diffusely growing tumor involved the right hypothalamus, right optic tract, basal leptomeninges, and floor of the 3rd ventricle. The pineal body was the size of a cherry and transformed into a solid tumor.

Histological diagnosis: Pinealoma.

Comment. This tumor clinically simulated a glioma of the optic chiasma and nerves. The tumor of the pineal body was distinctly separated from the neoplasms in the more rostral portions of the brain and was thought to represent the primary lesion, while those in the hypothalamus, optic chiasma, etc., were metastases.

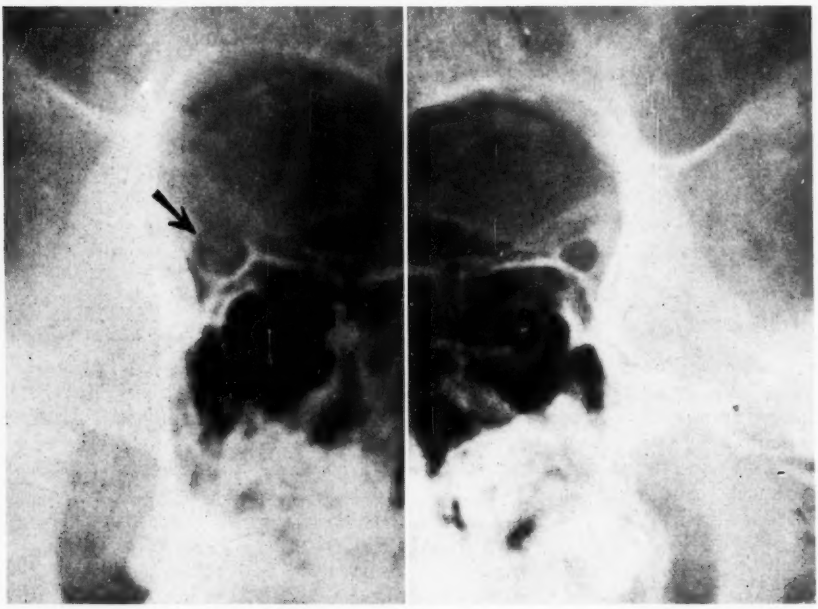

FIG. 5. *Case 2.* Roentgenograms of left and right optic foramina showing erosion and enlargement of left (arrow) optic foramen.

Case 3. D.M., a 15-year-old white female, was admitted on July 22, 1948. Five years previously she had had periods of increased thirst and urination which lasted for 2 months. In 1947 her vision and memory became progressively impaired and she complained of frequent periods of somnolence which often occurred during conversation. The patient had never menstruated.

Examination. She was small and slightly obese. She had neither pubic nor axillary hair and her breasts were poorly developed. There was early bilateral optic atrophy and a temporal hemianopsia on the right. The body temperature varied from 99 to 101°.

Specific gravity of urine was 1.020. CSF pressure was 130 mm. of water; total

protein 150 mg./100 cc.; colloidal gold curve 4222221000; mastic curve 555432; globulin 4+. Blood and CSF Kahn reactions were negative.

Roentgenograms of skull and pneumoencephalographic studies were inconclusive. An EEG indicated a deep anterior midline lesion of the brain.

Operation. On Aug. 10, 1948, a right frontal osteoplastic craniotomy was performed with removal of a bluish, firm tumor beneath the optic nerves which did not invade either the nerves or the chiasma. The neoplasm seemed to have been totally removed.

Course. Hyperthermia developed, and the patient died on the 4th postoperative day.

Autopsy. The brain weighed 1320 gm. There was a small blood clot in the region

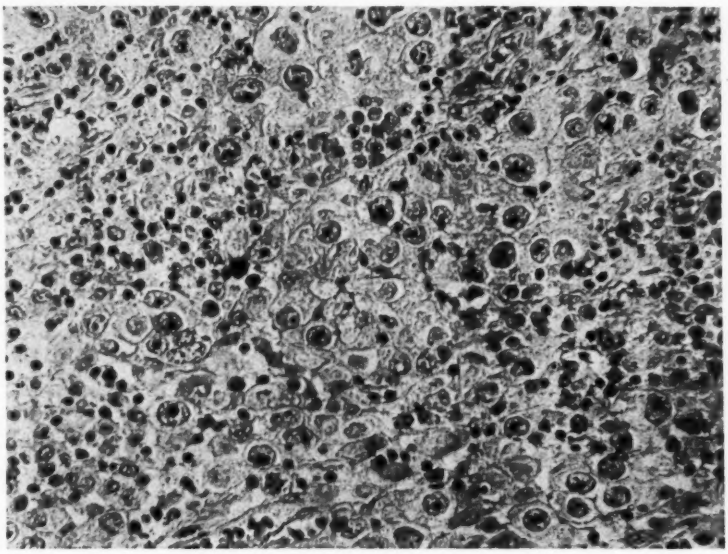

FIG. 6. *Case 3.* Photomicrograph of ectopic pinealoma.

of the optic chiasma and the hypothalamus. In the anterior part of the septum pellucidum there was a neoplasm approximately 1 cm. in diameter, but no remnants of tumor were present in the region of the optic chiasma. The pituitary gland was in its normal position. The pineal body was normal in appearance.

Microscopical Examination. The tumor removed at operation was histologically identical with that found in the septum pellucidum at autopsy and was characteristic of pinealoma (Fig. 6). The pineal body was calcified but was free of neoplasm.

Comment. The neoplasm, which was interpreted as a true ectopic pinealoma, was confined to the anterior hypothalamic region, which explains the occurrence of diabetes insipidus and hypogonadism. There is no satisfactory explanation for the narcolepsy.

Case 4. E.L., a 13-year-old white male, noticed defective vision of the left eye in

February, 1940. His vision continued to fail and 6 months later he became drowsy, had difficulty in walking, and complained of diplopia, headache and progressive deafness. One week before admission on Feb. 2, 1941, there developed hyperkinetic motion of all four extremities and a tremor of his left hand.

Examination. The patient was drowsy and mentally dull, with left divergent strabismus, loss of upward gaze and bilateral papilledema of 3 D. Visual fields were normal. He had mild nerve deafness bilaterally and a central 7th nerve palsy on the left. Reflexes were active and equal, with bilateral Babinski sign. He made continuous purposeless movements with his arms and legs. Secondary sexual characteristics had not developed.

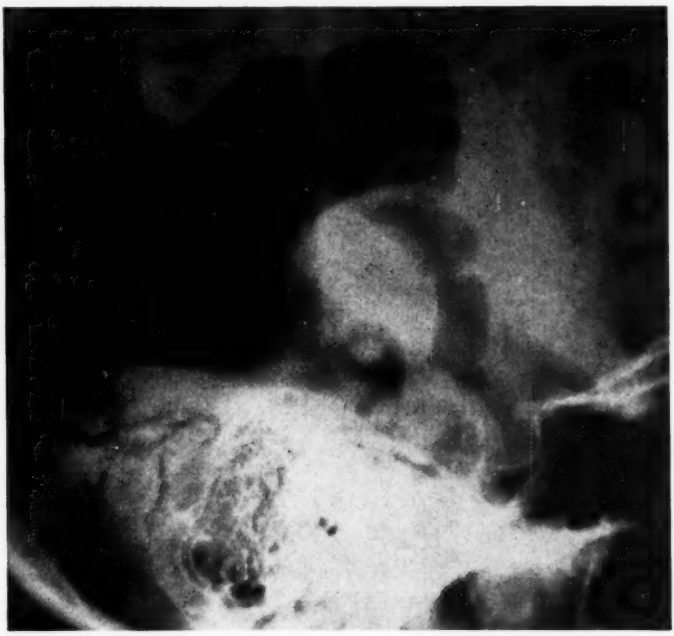

FIG. 7. *Case 4.* Ventriculogram showing tumor mass in posterior inferior margin of 3rd ventricle and inferior displacement of calcified pineal body.

Roentgenograms of the skull showed bilateral extrasellar erosion. There was downward and forward displacement of a densely calcified pineal body in the midsagittal plane. Ventriculograms showed that the lateral ventricles and the anterior portion of the 3rd ventricle were markedly dilated. A mass was located in the midline which involved the posterior inferior part of the 3rd ventricle and obstructed the aqueduct of Sylvius (Fig. 7). The 3rd ventricle and the pineal body were in the midline.

Operation. On Feb. 6, 1941, a right parieto-occipital craniotomy was performed and a large tumor was found which extended over the corpora quadrigemina and to the floor of the 3rd ventricle. The falx cerebri and the tentorium cerebelli were split and the neoplasm was completely removed by piecemeal.

Pathological Examination. The tumor tissue was fibrous and was calcified in its lower part. Histological diagnosis: Pinealoma (Fig. 8).

Postoperative course was relatively uneventful and the patient was discharged 16 days later.

Sixteen months after operation he complained of headache, vomiting, generalized weakness and anorexia of 4 months' duration. Examination revealed a left homonymous hemianopsia, secondary optic atrophy and divergent strabismus on the left. Babinski's sign was present bilaterally. Ventriculogram showed a tumor mass in

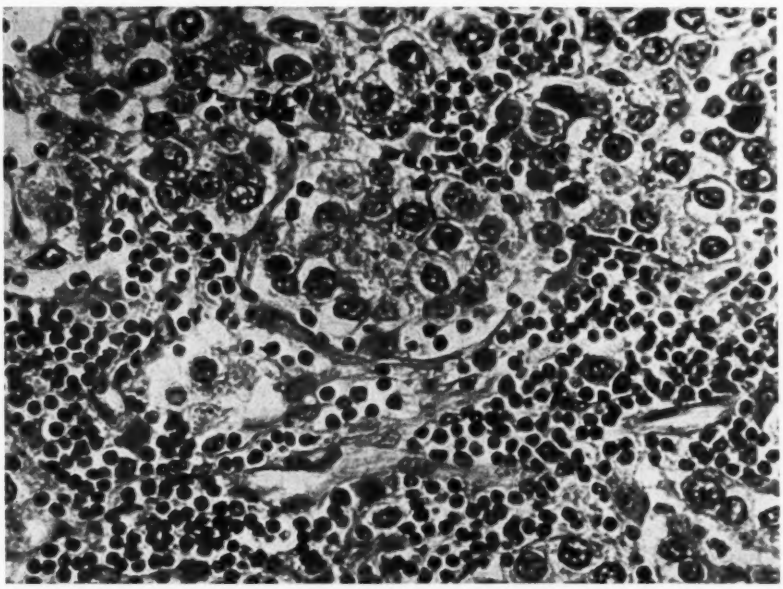

FIG. 8. *Case 4.* Photomicrograph of pinealoma.

the posterior portion of the 3rd ventricle which protruded to the left side and suggested a recurrence.

The patient was subsequently given irradiation therapy elsewhere (amount not known) with apparent improvement. Eleven years after operation and 10 years after irradiation he is living and, according to his physician, his condition is satisfactory. He has difficulty with fine coordination of his fingers and continues to have limitation of upward gaze.

TREATMENT

Direct surgical attack upon the neoplasm was performed by Dandy's approach[8] in 16 cases and by Van Wagenen's[25] method in 1. The operative mortality was 70 per cent. Only 5 patients survived for a period of 4 to 151 months. At the time of this writing only 1 patient is living (132 months) after the operation (Table 2).

Because of this exceedingly high postoperative mortality rate, a more conservative treatment was used in 12 cases. Third ventriculostomy[21] was

performed in 9 and ventriculocisternostomy[22] in 3 cases, followed by x-ray therapy. The present preferred method of roentgen therapy consists of administering 150–200 r to one field a day, using right and left lateral skull ports of 12×12 cm. over the pineal region to a total of 2000–3000 r in air to each field. (Technic: 200 Kv., ½ mm. Cu+1 mm. Al filtration, F.S.D. 50,

TABLE 2

Summary of treatment

Treatment	Pathologically Verified Cases		Non-Pathologically Verified Cases	
	No. Cases	Survival-Months	No. Cases	Survival-Months
Third ventriculostomy	1		9	
No irradiation therapy	1	0		
Irradiation therapy (measured in air)				
Less than 5000 r			1	139
5000–6000 r			5	126, 82, 64, 55, 23
More than 6000 r			3	87, 62, 16*
Torkildsen operation	0		3	
No irradiation therapy			0	
Irradiation therapy (measured in air)				
5000–6000 r			1	82
More than 6000 r			2	9,* 6*
Surgical removal	17		0	
No irradiation therapy	12	Immediate post-operative death in 11 cases 151*		
Pre-op. irradiation therapy	1	0		
Postop. irradiation therapy	2	50,* 132		
Pre-op. third ventriculostomy and pre-op irradiation therapy	1	4*		
Postop. third ventriculostomy and postop. irradiation therapy	1	11*		
Cases not treated	1	0	1	0
Total	19		13	

* Deceased at time of follow-up.

H.V.L. 1.0 mm.) One might rightfully conclude that the results of this treatment are much more favorable since 9 of these patients are alive and well 23 to 139 months after initial treatment (Table 2). In all patients who initially presented loss of upward and downward conjugate gaze, residual deficit remains. Only 2 of the 3 patients with symptoms that suggested seeding into the leptomeninges of the spinal cord had irradiation of the spinal cord axis. They survived for only 9 and 16 months.

DISCUSSION

The presence of tumors in the posterior portion of the 3rd ventricle, including neoplasms of the pineal body, is indicated by increased intracranial pressure and/or signs of involvement of regional anatomical structures. The latter have specific localizing value and include loss of upward conjugate deviation of the eyes (Parinaud's syndrome) and abnormalities of pupillary reflexes.

The size of the tumor may be suggested by certain clinical findings; loss of downward conjugate deviation of the eyes, impairment of hearing and cerebellar signs indicate a posterior growth. Inferior extension into the hypothalamus and midbrain is manifested by hypersomnia and precocious puberty. Diabetes insipidus follows involvement of the anterior part of the 3rd ventricle by direct growth or seeding. Pyramidal and thalamic symptoms occur when the neoplasm extends laterally.

The plain roentgenogram is often of great value. In 7 patients, 5 of whom were 15 years or younger, the tumor was suspected because of solid calcification of the pineal body; in 1 instance the pineal body was displaced inferiorly in the midsagittal plane (Fig. 7). The roentgenograms showed abnormal amounts of calcification in the pineal region in 5 patients who were beyond the age of 14. According to Camp,[3] calcification of the pineal in patients under the age of 10 is rare and "when present should suggest a pinealoma."

Calcification of the pineal or scattered calcification in this region, although important for clinical diagnosis and localization, does not reveal the histopathologic type of the neoplasm, since it may be present in various tumors such as teratoma, pinealoma, and pinealoblastoma. Although the roentgenographic appearance of centrally located calcification surrounded by a peripheral shadow of diminished density is indicative of pineal teratoma,[3] this finding was not present in the 4 teratomas in this series.

Ventriculography affords the most accurate localization of the tumors in the posterior portion of the 3rd ventricle. The degree of deformity observed in this ventricle is determined by the size and shape of the tumor. In general, the projecting contour of the neoplasm into the ventricle is convex, presenting either a smooth or an irregular outline. Some neoplasms may almost fill the 3rd ventricle and may partially obstruct the foramina of Monro. Since the tumors usually occupy a midline position, there is no lateral shifting of the 3rd ventricle. The larger tumors may produce some alteration of the floor and medial walls of the lateral ventricles. Although the roentgenogram and ventriculogram were very valuable for localization of the tumors in the present series, no correlation of the roentgenographic findings could be made with the histological structure.

Angiography was not used in any of these cases. According to Orley,[18] arteriograms demonstrate internal hydrocephalus, whereas the venograms show the vein of Galen to be displaced superiorly, thus losing its normal curve as it circumvents the splenium of the corpus callosum.

Krabbe[13] introduced the term pinealoma for neoplasms of the pineal body because of their microscopic similarity to the fetal structure. Bailey and Cushing[2] distinguished undifferentiated pineal tumors as pinealoblastomas. Russell[20] believed that tumors of the pineal body in reality represent a mixed tumor with pinealomatous and teratomatous features. This concept makes differentiation into various groups difficult. However, in the material available, the 4 teratomas were composed of tissues representing all three germinal layers and could be distinguished from pinealoma or pinealoblastoma without difficulty.

McLean[16] described a case of teratoma involving the posterior wall of the 3rd ventricle in which the pineal body was intact, and designated this lesion a parapineal teratoma. He suggested that such tumors originated from outpouchings of the diencephalon other than the pineal. Similar conditions may also apply to pinealomas. A case of parapineal pinealoma which apparently originated from the posterior wall of the 3rd ventricle beneath an intact pineal body is among the present series.

Troland and Brown[24] and others designated as "ectopic" those pinealomas that arose primarily in the anterior part of the 3rd ventricle and had no demonstrable connection with the pineal body. Recognition of true "ectopic" pinealoma depends on postmortem examination since operative disclosure of a pinealomatous tumor in the anterior part of the 3rd ventricle cannot distinguish it from seeding. This is illustrated by our Cases 2 and 3; in the latter the tumor was a true ectopic pinealoma and in the former the pinealoma was pseudo-ectopic.

Direct surgical removal of tumors of the pineal region is not recommended because of the high mortality rate entailed. When this was attempted, very few patients survived the operation for an appreciable length of time (Table 2). As can be observed in reviewing this material and from the experience of others[11,12,23] third ventriculostomy or ventriculocisternostomy in combination with x-ray therapy is the preferred form of treatment giving the individual patient maximum chance of survival with minimal neurological deficit. Surgical extirpation of the neoplasm should not be considered in a patient for whom conservative therapy has not been adequately carried out.

In the cases in which the patients were treated for obstructive hydrocephalus by third ventriculostomy or ventriculocisternostomy, only a presumptive diagnosis of tumor can be made by clinical and ventriculographic findings. The true histological structure and correlation with growth behavior of the tumor remain obscure. In this region certain neoplasms are radiosensitive as shown by Case 4, in which a patient with a pinealoma presented signs of recurrence following surgical removal and later demonstrated a marked clinical improvement after irradiation therapy. On the other hand, some tumors may show little or no growth, as seen in Torkildsen's patients 1 and 4[23] who initially presented only signs of increased intracranial pressure and in whom ventriculography showed no appreciable change in the size of

the neoplastic mass 5 and 7 years after ventriculocisternostomy without x-ray therapy. While it may be justifiable[15] to defer irradiation therapy in such cases until there are clinical signs of involvement of regional anatomical structures, we believe the majority of patients should be given immediate roentgen therapy.

SUMMARY

1. Thirty-two cases of tumors of the posterior portion of the 3rd ventricle and 1 case of true ectopic pinealoma in the anterior part of the 3rd ventricle are presented.

2. An anatomical explanation for clinical symptomatology is reviewed.

3. Although roentgenographic studies afford accurate localization of tumors in the pineal region, it was not possible in this material to correlate the histological structure with the type of calcification observed in the pineal region or the appearance of the tumor mass as outlined by ventriculograms.

4. Conservative treatment offers the patient a maximum survival period with minimal neurological deficit.

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PAINFUL SPINA BIFIDA OCCULTA

WITH REVIEW OF THE LITERATURE

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IN A review of the literature from 1930 to 1950, 14 references to painful spina bifida occulta were found.[1–3,5–7,9,11,12,14,21,24,32,34] Usually it is thought that spina bifida occulta is of no clinical significance and that it produces no symptoms, but actually a certain percentage of cases of low-back pain and even of pain that extends into the extremities are attributable to this congenital defect. It is known that myelodysplasia and filum terminale traction may cause organic changes in the lower extremities.[26]

We were able to find 48, or 0.8 per cent, out of a group of approximately 6,000 cases of low-back pain, in which we thought the pain was produced by spina bifida occulta. This conclusion was arrived at mainly by a process of exclusion of the usual entities known to produce low-back pain, and then only after a series of examinations and re-examinations and a review of these cases over and over again. We have operated upon 18 patients in this group and have obtained very gratifying results. Therefore, we thought this lesion should be more thoroughly studied and evaluated and brought to the attention of those dealing with low-back pain.

HISTORICAL CONSIDERATION

In 1641 Nicholas Tulp, a Dutch physician, first described spina bifida and by 1652 he had reported 6 cases.[29] Virchow[30] in 1875 was first to describe spina bifida occulta. Then followed a group of clinical reports by von Recklinghausen[25] in 1886, Denucé[4] in 1906, Hesse[17] in 1918, Sternberg[27] in 1929 and many others,

Up until 1915, 85 cases of spina bifida occulta had been reported. With the development of better X-ray equipment and its more general usage, it was soon found that the lesion was quite common. At the present time it is thought to occur in the low back in about 3 per cent of the adult population.

Next followed many papers explaining the faulty embryological development.[8,10,19,20,22,25,33] The earlier theories of causation are of interest. Lebedeff[22] in 1881 felt that the lesion was caused by the bending backward of the embryo in utero. This idea of stress on the neural plate preventing closure of the neural tube was also supported by Ernst[8] in 1909. von Recklinghausen[25] in 1886 thought the defect was the result of incomplete development of mesodermal tissue. Frazier[10] in 1918 suggested that increased spinal fluid pressure, from delayed absorption, was the causative mechanism. Sachs[13] in 1934 reported that hyperactivity of the choroid plexus in utero might cause the condition.

Let us consider some underlying physiological or biochemical disturbance as the causative mechanism for spina bifida occulta. Levine[23] in 1942 presented the isosensitization theory as being associated with congenital defects. Further elaboration on this theory was made by Wiener[31] in 1947. Many have accepted the present-day concept of the antigen-antibody reaction as a primary cause of developmental defect. In this concept the antibodies from the mother pass through the placenta into the fetus, producing certain types of physiological reactions, since there is an isosensitization of the mother to the antigens from the fetus. This may produce (1) intravascular hemolysis, or (2) intravascular coagulation (clumping of fetal erythrocytes). We wish to suggest that this process may produce the embryological developmental defect known as spina bifida occulta.

PHYSIOPATHOLOGY

The relationship of low-back pain to spina bifida occulta is clear when the physiopathological nature of the lesion is understood. With the incomplete development of the neural arch, nature fills the defect with dense fibrous tissue, which sometimes encases the dura mater snugly. The ligaments in the area are not attached to a true osseous structure but to this fibrous-tissue replacement. This fibrous mass, being closely associated with the dura and the nerve roots, can effect a stretching or compression syndrome producing local or referred pain.

Our interpretation of the gross pathological lesion in spina bifida occulta producing pain, agrees with observations made elsewhere in the literature.[3,5,6,9,11] There is an inelastic, unyielding fixed mass of fibrous tissue filling the space normally occupied by the midline structures of the low back. The usual mechanics of motion are altered. Normally, the dura is free and protected by the epidural fat with ample space about it. The laminae and the posterior spinous processes serve as additional protection to the dural canal and spinal nerves. Traction of ligaments, muscle pull, stress and strain, are absorbed by these rigid structures. But when the latter are absent, this force is transmitted to the dural canal and spinal nerves of the area involved. A mass of fixed and even hard fibrous tissue extends from the superficial fascia to the dura. This mass of tissue may be quite large, depending upon the size of the defect. Sometimes it practically encases the thin poorly developed dura mater and the nerve roots emerging. This sort of pathological structure does not serve to protect the dura and nerves as well as does the normal bony canal with epidural fat in place. It does not permit motion without traction. Instead, bending and twisting cause direct dural and nerve pull and this in turn produces pain. This same condition is often produced by postoperative scar tissue, as has been demonstrated at re-operation for discs or after a laminectomy for other spinal cord pathology.

In 3 instances we have also observed impingement upon the canal by nubbins of misshapen bone representing the rudimentary lamina. These pieces of bone may project into the canal from their lateral attachment and

produce dural compression with indirect nerve involvement. Or, as in the 3 cases noted, the rudimentary lamina may directly impinge upon the nerve root extradurally and cause pain.

Since the above physiopathological processes have occurred repeatedly in our cases, we feel they are a reasonable explanation of the low-back pain in spina bifida occulta.

SYMPTOMS

In most cases symptoms and signs are completely absent. Occasionally low-back pain will be the only complaint. Hadley[16] reported that 4.1 per cent of individuals with spina bifida occulta have low-back pain. This pain may vary in degree, location and extent. It depends, however, on the anatomical structures or tissues involved and the manner in which the pain is produced.[5] Sensations of heaviness, and pulling of the back muscles on moving or bending[11] may occur with local back pain. With involvement of the lumbar nerves there may also be radiation of pain into the buttocks and hamstring muscles.[28]

Local inspection often reveals a subcutaneous lipoma,[15,18] a telangiectasis or a dimple over the lumbar or sacral region, that may suggest the underlying defect. There may be an ironed-out lumbar lordosis associated with local tenderness over the back muscles. Reflexes in the lower extremities are sometimes diminished. There may or may not be a positive Lasègue's sign. Other and more marked findings, such as motor and sensory changes, with deformity of extremities, hypertrichosis, etc., occur with the more extensive types of spina bifida occulta.

Dittrich[5] reported 5 cases in which the patients required absolute bed rest for 3 weeks or more because of the intense low-back pain. A laminectomy with removal of the pathological structures resulted in complete relief of the pain.

CASE REPORTS

The following cases may serve to illustrate the syndrome of painful spina bifida occulta.

Case 1. F., a woman 41 years of age, was first seen by us in December 1940. She had been disabled for the preceding 3 weeks because of pain in the low back, extending into the buttocks on both sides but never into either lower extremity. During the past 4 years, pain had been intermittent and caused considerable suffering. Bending and twisting would aggravate it. A pelvic operation for malposition of the uterus had been performed in 1936. The low-back pain continued.

Examination. Reflexes in the lower extremities were present and equal. There were no sensory or motor changes. The sphincters were normal. A fat pad was palpable over the sacrum and a defect in the spine could be felt beneath it. X-rays showed that the lamina of the 5th lumbar vertebra was not fused. There was a defect in the posterior arches of the 1st and 2nd sacral segments (Fig. 1).

Operation. On Jan. 14, 1941 a large fibrolipomatous pad extending from the superficial fascia to the dura was disclosed (Fig. 2). The dura was firmly encased by this structure while the external portion of the fibrolipomatous pad became continu-

ous with the lumbar fascia. It was necessary to dissect this fibrous-tissue encasement from the dura throughout the extent of the long defect. The dura was much thinner than normal.

Course. Convalescence was normal. The patient has remained free of pain to date, 11 years later.

Case 2. R.K., a male aged 40 years, was seen first in February 1946. This man was a tense business executive and had been suffering from pain in the low back for 12 years. At times the pain was in one leg and then the other. He had had numerous examinations in various clinics and had been studied thoroughly from all angles. X-rays and other studies were repeated over and over again and always the patient

FIG. 1. *Case 1.* Roentgenogram showing congenital bony defect in posterior arches of 1st and 2nd sacral segments.

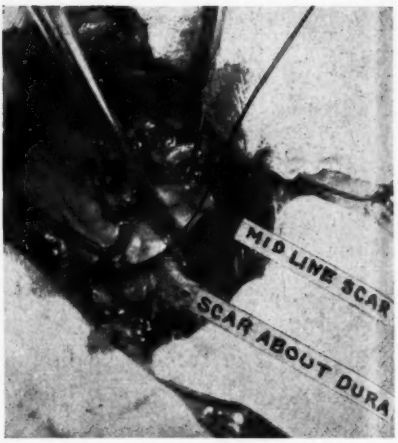

FIG. 2. *Case 1.* Midline scar and scar about dura mater disclosed at operation.

was told that nothing could be found. The low-back pain continued to cause considerable suffering that tended to incapacitate him about half the time. He became discouraged as the years slowly passed without relief. Every now and then he made a new effort to secure relief and would go to another medical center for another complete study. Fortunately he was well able to afford these numerous and extensive medical studies. Finally he resorted to osteopathic treatment, a low-back brace, and empirin compound as a daily routine.

Examination. He was a large, well developed individual. BP was 160/115. His back was straight. There was tenderness on pressure over L5 and in the midline, and over the upper part of the sacrum. All reflexes were normal. There was no hypesthesia over the lower extremities and there were no sphincteric changes. Bending would cause pain in the back. Lying down would give relief.

X-rays showed an incomplete fusion of the posterior arch of the 1st sacral segment and a very small L5 lamina. There was considerable lack of bony development in the S1 and L5 segments (Fig. 3).

Clinical Diagnosis. Considering this patient from an objective standpoint, there was very little with which one could hope to explain his troubles. Organically, the congenital defect in the lower spine was all one could find. Since the defect was in the exact site of his pain, and in view of our Case 1, we talked the matter over with the patient. We explained that again we found nothing organically wrong with him except the spinal defect which it had been our custom in previous patients to discount entirely, with one exception. This woman (Case 1), with a similar history and similar findings, had been operated upon and had done well. The patient, being an

FIG. 3. *Case 2.* Roentgenogram showing bony defect in S1 and L5 segments.

FIG. 4. *Case 2.* Fibrous tissue extending from lumbar fascia to dura mater.

astute business man, thought the matter over carefully and reported back for surgery.

Operation. On April 8, 1946, under local anesthesia, an incision was made from L4 to S2. A midline fascial septum extended down to a fibrous-tissue mass which filled the space ordinarily occupied by the posterior spinous process and the lamina of the 1st sacral segment (Fig. 4). All the scar tissue and fascia were dissected free from the dura. The 4th and 5th interspaces were explored with a nerve hook and nothing could be found in the way of a disc protrusion; in fact the operator could put his finger in the defect of the lamina and palpate the anterior surface of the canal.

Course. Convalescence was normal, and the patient has had no pain subsequently. He is very gratified over the relief he has obtained, and has asked repeatedly why he had not been operated upon long before.

Case 3. M.H., a farmer aged 38 years, complained of low-back pain extending into both hips, for the past 20 years. He had been disabled for 4 to 5 weeks at a time

on several occasions. Coughing and sneezing did not aggravate the pain, and lying down relieved it. Bending, twisting and turning caused pain.

Examination. There was tenderness on pressure over L5 on both sides and over the sacrum. There was no visible change in this area, but a bony defect could be palpated. Roentgenograms showed a spina bifida of the 5th lumbar and 1st sacral segments (Fig. 5). The lumbosacral angle was increased.

Course. We followed this patient for almost a year. Palliative measures, such as physiotherapy, medication and a brace did not give satisfactory relief.

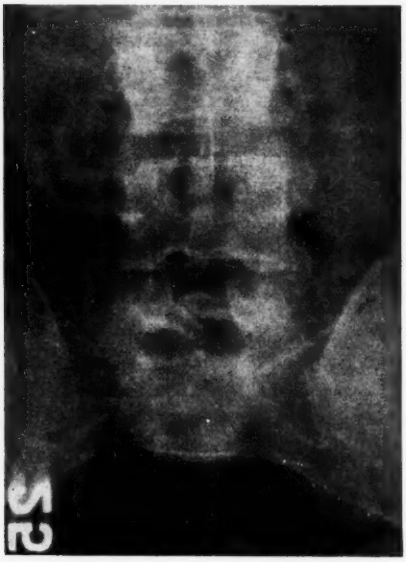

FIG. 6. *Case 3.* Scar tissue removed at operation.

FIG. 5. *Case 3.* Bony defect of 5th lumbar and 1st sacral segments, with curved laminal remnant compressing S1 root.

Operation was performed on Oct. 30, 1945. The fascia extended into the midline all the way down to the lamina and about the edges of the bony defect. What seemed to be dura was actually a fibrous encasement of the dura. When this fibrous encasement was opened, epidural fat was found beneath it. The dura was dissected free. The dura and cauda equina were definitely compressed and constricted by this tissue (Fig. 6). It was noted that the undersurface of the 5th lamina remnant compressed S1 nerve root. This was rongeured away.

Histological diagnosis: Hyalinized connective tissue.

Course. Convalescence was normal, and the patient returned to his duties as a farmer. He has not complained of any more trouble.

Case 4. A.K., a male aged 47 years, complained of intermittent low-back pain extending into the buttocks but not extending into either lower extremity for the past 20 years. He had been incapacitated at times, but for 2 weeks prior to admission in August 1948, the pain had been constant.

Examination. There was tenderness on pressure over L5 and the upper sacral region, more in the midline than elsewhere. Reflexes were normal. There were no sensory or motor changes, and no sphincteric changes. Roentgenograms of the

lumbosacral spine and pelvis showed slight reduction in the normal lumbar lordosis, possibly associated with some muscle spasm. The intervertebral joint spaces were normal. There was a large defect in the posterior arches of the 5th lumbar and 1st sacral segments (Fig. 7), a developmental variation that frequently is not associated with clinical symptoms.

Operation, Sept. 4, 1948. It was found that the nubbin of the right L5 lamina was freely movable, and this was removed. The fibrous lipomatous mass filling the bony defect was carefully resected, including that part encasing the dura.

Course. Recovery was normal. The patient has had no more complaints and has continued his work as a dairy salesman.

FIG. 7. *Case 4.* Bony defect of posterior arches of L5 and S1 segments.

COMMENT

The above cases would indicate that low-back pain, with extension of pain into the hips, is rather characteristic of this condition. Sometimes, however, the pain may extend into one or both lower extremities. The fact that pain is on both sides is certainly significant. Also, there is a history of recurrent and often continuous pain over a number of years, aggravated particularly by bending. Usually there are no reflex changes or sensory changes.

These people seem to represent a group of spina bifida occulta who have not had the additional cord changes as occur in the more severe cases, or when myelodysplasia is present. We have not noted any cord involvement through traction by the filum terminale.

It seems that the mechanism producing the pain is caused by nature's effort to fill in the bony defect with a fibrous or fibrolipomatous tissue, and there is often found a constricting mass of fibrous tissue involving the dura mater and at times the sheaths of the emerging roots in the area.

Our interpretation of the gross pathological lesion in this condition is based on the premise that painful spina bifida occulta occurs usually in those individuals with a minimal bony defect who have no myelodysplasia or signs of cord involvement. In the cases in which we have performed operation, there was a traction mechanism produced by dense fibrous tissue, which many times encased and constricted the dura mater, and sometimes even produced traction on the nerve roots. The fibrous tissue extended from the dura all the way up through the spaces where ordinarily the posterior spinous processes would have developed, and attached itself to the lumbar fascia

of the back. In fact, the lumbar fascia invaginated in the midline and actually became part of the lesion, so that when the back was bent, traction was produced by the non-elastic fibrous-tissue structures. Sometimes small bands or cords of fibrous tissue could be found extending from the defect in the lamina to the dura mater and to the sheaths of the nerve roots. Occasionally lipomatous pads were interposed between the dura and the dense fibrous tissue encircling it, but this was not a usual finding.

Microscopically, local swelling with minimal hemorrhages of the tissue was noted. There were some calcified flakes in the connective tissue, indicating old hemorrhagic areas. Some lymphocytic infiltration was noted. In one case there was hyalinization of the connective tissue.

SUMMARY

1. A review of the literature on spina bifida occulta is presented.

2. The possibility of isosensitization between mother and fetus is suggested as a causative mechanism for this developmental defect.

3. Low-back pain and pain into the lower extremities may occur with spina bifida occulta. The mechanics of this pain are explained.

4. The symptomatology of painful spina bifida occulta is given, with illustrative case reports.

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STUDIES ON CEREBRAL INTRAVASCULAR PRESSURE

FURTHER DATA CONCERNING RESIDUAL PRESSURE IN COMPONENTS OF THE CIRCLE OF WILLIS AND IN BLIND ARTERIAL SEGMENTS*

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OBJECTIVE measurements of intravascular pressure within the carotid vessels of the neck by means of differing techniques have been reported by Sweet and Bennett,[5] Sweet, Sarnoff and Bakay[6] and by Brackett and Mount.[3] Stimulated by these studies, efforts have been made to extend these observations to a recording of intravascular pressures in components of the anterior portion of the circle of Willis and in the cortical arterial vessels of vascular anomalies of the brain. The safety and technical feasibility of recording intravascular pressure by the introduction of a #27 needle into the anterior cerebral, middle cerebral and internal carotid arteries of the brain in patients with vascular lesions of the nondominant hemisphere have been published,[2] with a detailed description of the amplifying and recording apparatus. A second report has furnished further data concerning intravascular pressure within these components of the circle of Willis with and without temporary proximal occlusion.[8] It has in particular described the development of serial, objective testing for the presence of adequate or inadequate cross-hemisphere circulation during intracranial operative procedures upon patients with proved congenital cerebral aneurysms or vascular anomalies of the brain.

This report is concerned with further data obtained from the recording of intravascular pressure in arterial vessels in neck and brain during operative procedures upon 25 patients with proved congenital cerebral aneurysms or vascular anomalies of the brain. Diagnostic arteriograms were obtained by the percutaneous method. At operation, under intratracheal balanced anesthesia, the anterior cervical triangle and the involved hemisphere were prepared and draped as a single operative field and serial observations were made concerning intravascular pressure as previously described.[8] Since an automatic flushing device was not available during these studies, such as that used by Bakay and Sweet,[1] minimal damping of systolic pressure peaks was unavoidable. The following data pertain to pressure readings in the carotid arterial tree, obtained in 17 of these patients. In the remaining 8 patients, intravascular pressure readings were restricted to the vessels of the neck.

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Eight of these 25 patients demonstrated percentage systolic pressure falls of 50 per cent or above when the intravascular pressure was measured in the internal carotid artery of the neck and this vessel was temporarily occluded at a point proximal to the recording needle. In 4 of these 8 patients, this marked degree of intravascular pressure reduction was verified by measurement of intravascular pressure in terminal intracranial vessels. In 2 of these patients, with percentage systolic falls above 50 per cent, clip ligation of the internal carotid artery intracranially, forced by rupture of the aneurysm,

TABLE 1

Systolic pressure percentage falls in carotid arterial tree following temporary occlusion of neck vessels

Case	Occlusion	Internal Carotid Artery Neck			Common Carotid Artery Neck		
	Percentage Systolic Pressure Fall in—	Internal Carotid Neck	Internal Carotid Brain	Middle Cerebral	Internal Carotid Neck	Internal Carotid Brain	Middle Cerebral
1. Vas. anomaly		50		37	44		40
2. Vas. anomaly*		28	29	34	30		37
3. Vas. anomaly					52	46	45
4. Vas. anomaly		47	47	48	47	56	
5. Cong. aneurysm		28	24	15†			
6. Cong. aneurysm		62	59	60	60	55	56
7. Cong. aneurysm		33	28	29			
8. Cong. aneurysm		79	84				
9. Cong. aneurysm		40	35	44	52	46	45
10. Cong. aneurysm		56		60	45		42
11. Cong. aneurysm		26		24	32		35
12. Cong. aneurysm		78	76	76	57	62	54
13. Cong. aneurysm		63	55	66	62	57	62
14. Cong. aneurysm		51	51				
15. Cong. aneurysm		31	33	36	36	38	
16. Cong. aneurysm		38	29	35	31	35	30
17. Cong. aneurysm		74		63	74		80

* Mean pressures.
† Reading made after clip ligation, anterior cerebral artery.

resulted in a residual intravascular pressure in the middle cerebral artery of less than 50 mm. of mercury (15 and 40 mm. of Hg respectively).[8] At this level of reduced intravascular pressure, hemiplegia and exitus resulted. With our present knowledge, controlled partial ligation of the appropriate neck vessel appears indicated in such cases.

A general consideration of these measurements suggests that intravascular pressure falls recorded in the internal carotid artery of the neck following temporary occlusion of this vessel proximally are repeated in degree both in the internal carotid artery intracranially and in the middle cerebral artery. Review of Table 1 indicates the marked variation in individual cases. The quantitative extent of reduction in intravascular pressure does not suggest that neck carotid ligation can represent an adequate solution to the per-

manent control of hemorrhage from a congenital cerebral aneurysm. The variable recordings in Cases 1 and 5 cannot be adequately evaluated.

The second group of pressure recordings to be detailed in this report have to do with the residual intravascular pressure found in blind or trapped carotid arterial segments. They fall into three groups:

(A) Those patients in whom a permanent blind or trapped carotid arterial segment was formed by complete ligation of the internal carotid artery in the neck and by clip ligation of the intracranial portion of this artery distal to the ophthalmic artery.

(B) Those patients in whom a similar but temporary trapped segment was formed by clip ligation of the internal carotid artery in the brain and temporary occlusion of this vessel in the neck.

(C) Those patients in whom trapped segments were formed in the intracranial portion of the internal carotid artery by clip isolation of an aneurysm.

Interest in this phase of the study was initiated by the finding of blindness in the ipsilateral eye immediately following procedures such as that noted in (A) in 2 patients in an earlier group of patients with congenital cerebral aneurysm.[4]

A brief description of individual patients and the pressure recordings follow.

GROUP A

V. W. Vascular anomaly. Ligation of internal carotid artery, neck: Clip ligation of internal carotid artery, brain.

1. Residual intravascular pressure in trapped or blind arterial segment — $\frac{60}{48}$

2. Occlusion, external carotid artery — $\frac{60}{48}$ to $\frac{54}{44}$

3. Repeat — $\frac{60}{48}$ to $\frac{52}{44}$

Clinical result: Died of recurrent subarachnoid hemorrhage, 4th postoperative day.

J. H. Ligation of internal carotid artery, neck: Clip ligation of internal carotid artery, brain, for aneurysm of this vessel—$5\frac{1}{2}$ months after partial ligation, common carotid artery, neck.

1. Residual intravascular pressure, middle cerebral artery — $\frac{76}{44}$

2. Procedure as noted above. Residual intravascular pressure in trapped or blind arterial segment — $\frac{88}{40}$ to $\frac{44}{36}$ 50%

Clinical result: Excellent.

A. P. Ligation of internal carotid artery, neck: Clip ligation internal carotid artery, brain, for intracavernous arterial aneurysm.

1. Pre-ligation pressure readings in middle cerebral artery

a. Occlusion, internal carotid artery, neck	90/70 to	58/52	36%
b. Repeat	90/70	60/54	
c. Occlusion, common carotid artery, neck	90/70 to	44/40	51%
d. Occlusion, internal carotid artery, brain	80/64 to	56/50	30%

2. Ligature procedure as noted
3. Systolic pressure readings in trapped or blind arterial segment

a. Residual pressure	90		
b. Occlusion, external carotid artery	90 to	56	38%
c. Repeat	80 to	48	40%

Clinical result: Poor. Hemiparesis. Cause, arteriosclerosis?

GROUP B

G. J. Aneurysm of posterior communicating artery. Intravascular pressure readings in temporary trapped or blind arterial segment.

1. Clip ligation of internal carotid artery, brain, and neck of aneurysm of posterior communicating artery
2. Temporary occlusion of common carotid artery
3. Intravascular pressure in blind arterial segment 102/76 to 46/44

Clinical result: Transient hemiparesis on 2nd postoperative day. Otherwise excellent.

J. F. Aneurysm of anterior communicating artery. Intravascular pressure readings in temporary trapped or blind arterial segment.

1. Intravascular pressure readings in internal carotid artery, brain

a. Occlusion, internal carotid artery, neck	86/70 to	42	51%
b. Repeat—mean pressures	88 to	48	45%
c. Occlusion, internal carotid, brain, distal to reading point	42 to	52/48	12% *increase*

d. Release distal occlusion — 52/48 to 42

e. Release internal carotid artery, neck — 42 to 82/68

Clinical result: Excellent—intracranial trap ligation.

L. J. Aneurysm of posterior communicating artery. Intracranial clip ligation and isolation of aneurysm. Pressure in temporary blind arterial segment.

1. Pre-ligation intravascular pressure in middle cerebral artery with temporary occlusion of internal carotid artery, neck — 98/78 to 74/62 — 24%
2. Procedure as noted above
3. Residual post-ligation intravascular pressure, middle cerebral artery — 64/44
4. Intravascular pressure in internal carotid artery, brain, proximal to clips with temporary occlusion, internal carotid artery, neck — 102/78 to 60/56 — 41%
5. Same, with temporary occlusion, common carotid artery — 96/78 to 40 — 58%

Clinical result: Excellent.

GROUP C

J. J. Aneurysm of posterior communicating artery. Intravascular pressure readings in intracranial blind arterial segment, between clips isolating aneurysm.

1. Pre-ligation intravascular pressure readings in middle cerebral artery
   a. Occlusion, common carotid artery — 60/40 to 42/32 — 30%
   b. Occlusion, internal carotid artery, neck — 62/38 to 40/30 — 35%
2. Clip ligation or isolation of aneurysm
3. Intravascular residual pressure in internal carotid artery, brain, between clips — 48
4. Clip ligation of base of aneurysm

Clinical result: Temporary mild hemiparesis on 3rd postoperative day. Otherwise excellent.

R. P. Aneurysm of posterior communicating artery. Intravascular pressure readings in intracranial blind arterial segment, between clips isolating aneurysm. Normal pre-ligation pressure fall, middle cerebral artery.

1. Ligation of internal carotid artery, neck, and clip ligation of internal carotid artery, brain, distal to aneurysm of posterior communicating artery

2. Intravascular pressure in internal carotid artery, brain, proximal to aneurysm — 82 / 54
3. Clip ligation, internal carotid artery, brain, proximal to aneurysm
   a. Intravascular pressure in blind arterial segment between neck ligation and proximal intracranial clip — 48 / 44
   b. Intravascular pressure in internal carotid artery, between clips. Note increase in pressure as compared to pre-ligation reading — 104 / 58
   c. Intravascular pressure in internal carotid artery, brain, between clips, following ligation of feeder vessel, one of two, identity? — 104 / 58 to 76 / 58
4. Final clip ligation of neck of aneurysm

Clinical result: Excellent.

These recordings in Groups A and B show a uniform intravascular pressure fall to a level of approximately 50 mm. of Hg in the majority of patients. In Case A.P., the intravascular pressure remained high in the closed segment when the internal carotid artery was occluded but fell with common or external carotid occlusion. In another patient, not of this series, the intravascular pressure in the isolated carotid segment fell acutely to a level that could not be measured with technical surety. Although the recording data were perhaps not valid, it is interesting that visual acuity fell from 20/40 to 20/400 in the ipsilateral eye postoperatively.

The data recorded in group C appear quite significant since, in these 2 cases at least, simple isolation of the aneurysm did not reduce the intra-aneurysm pressure to a point where a clinical cure could be assured.

SUMMARY

Further data have been presented concerning the intravascular pressure in the intact carotid arterial tree and in isolated carotid segments. The clinical significance of these findings has been briefly discussed.

These data are of further interest in the light of Wolf's[7] discussion of hemodynamics as they may apply to a sacculated segment of a blood vessel, i.e., an aneurysm. Bernoulli's principle states that along a line of flow in a tube the lateral pressure is increased in a dilated portion where the velocity of flow is reduced but the percentage increase in lateral pressure must be quite small. On the other hand, Wolf points out that tension in a dilated wall, according to Laplace's formula, tends to increase in proportion to the radius of curvature of the sacculation. Thus rather marked increases in tension may occur in the walls of aneurysms with negligible changes in intravascular pressure and possibly at any level of intravascular pressure. These considerations would suggest that therapeutic efforts should be re-

oriented toward the tension factor rather than that of reduction of intravascular pressure alone. Significant relief of tension can be accomplished only by isolation or destruction of the aneurysm or by increasing the resistance of the aneurysmal wall itself to tension.

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THE ANEURYSMAL ORIGIN OF NONFATAL SUBARACHNOID HEMORRHAGE

AN ANGIOGRAPHIC SURVEY OF 53 CASES

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In 1947, an analysis was reported[1] of 130 cases of spontaneous subarachnoid hemorrhage found in the records of the Buffalo General Hospital between the years of 1929 and 1945, inclusive. The cause of the bleeding was determined at autopsy or at operation in 47 cases. In 44 cases (93.6 per cent), the bleeding originated from intracranial aneurysms. The statement was made, "It seems reasonable to assume that in the unproved cases, the percentage due to aneurysms would parallel that of the proved cases." Hyland[2] also said that "the accumulated evidence . . . indicates a sufficiently high incidence of demonstrable aneurysms as the cause of this clinical syndrome to warrant consideration of the prognosis in unverified cases in terms of aneurysm." Martland[3] found that practically all cases of fatal subarachnoid hemorrhage are caused by rupture of intracranial aneurysms. He felt, however, that most of the cases of spontaneous subarachnoid hemorrhage with recovery (roughly 50 per cent) "are probably not due to 'leaking' or ruptured intracranial aneurysms, but rather to some unexplained infection or to some as yet unknown cause."

Since our earlier report, cerebral angiography has been employed with increasing frequency in diagnosis of cases of subarachnoid hemorrhage not immediately fatal. It was considered interesting to determine to what degree this adjuvant was helpful in the diagnosis of subarachnoid hemorrhage.

In 324 patients subjected to cerebral angiography in the Buffalo General Hospital between 1947 and 1951, inclusive, were 53 in whom the test was made for spontaneous subarachnoid hemorrhage. Unfortunately, it was not at first realized that, to be as conclusive as possible, the entire intracerebral vascular tree must be visualized. The 53 tests were done by injection of both carotids and a vertebral artery in only 3 cases, of both carotids in 22, of 1 carotid in 26, of a single vertebral artery in 1 case, and in 1, no successful vascular visualization was obtained. In cases in which only one carotid artery was injected, the test was made on the side suggested by the patient's clinical signs.

In the 53 cases of subarachnoid hemorrhage, angiograms disclosed aneurysms in 22 cases (41 per cent) and an arteriovenous malformation in 1 case. The aneurysms were on the carotid artery in 7 cases, carotid bifurcation (anterior cerebral-middle cerebral branching) in 2, anterior cerebral in 6, anterior communicating in 2, middle cerebral in 4 and several vessels were

involved in 1 case. In 4 other cases the angiograms did not indicate aneurysms that were found later at autopsy. These were on the posterior communicating artery in 1 case, and on the anterior cerebral-communicating complex in 3. In another case a unilateral carotid injection revealed 2 aneurysms on the left middle cerebral artery, and a third was found on the contralateral middle cerebral artery at autopsy.

In the 27 cases of aneurysms verified to date, 4 (15 per cent) were missed by angiography as it was employed in this series. It obviously is not possible to determine in how many other of the remaining 26 patients with subarachnoid hemorrhage, aneurysms were missed in like manner.

In the group of 27 patients having proved aneurysms, 10 of the angiograms were performed with bilateral and 17 with unilateral carotid injections. Three of the 4 unvisualized aneurysms were found in patients who had had bilateral injections. In the fourth case the injection was made on the side on which the proximal anterior cerebral artery was congenitally absent; the aneurysm was on the anterior communicating artery irrigated by the uninjected carotid. Three of the 4 unvisualized aneurysms involved the anterior communicating artery. The fourth was found at autopsy embedded in a cerebral peduncle, but its involved vessel was not identified. In this case both carotids and a vertebral artery were injected for angiography.

DISCUSSION

In a series of patients surviving subarachnoid hemorrhage, a smaller percentage of aneurysms (41 per cent) was disclosed by angiography than in a series studied at autopsy (93.6 per cent).

While other factors may be involved in the production of spontaneous subarachnoid hemorrhage, aneurysms can be demonstrated to be responsible for the bleeding in a high percentage of the patients who recover from a given episode, as well as in the fatal cases.

In this series subarachnoid hemorrhage was caused by aneurysms in 26 cases and by an arteriovenous malformation in 1 case.

Despite its shortcomings and its imperfect utilization in this series, angiography is the best adjuvant available at present for identification of the lesions responsible for subarachnoid hemorrhage.

Visualization of the entire intracranial vascular tree is necessary to disclose the largest possible number of aneurysms.

Even complete angiography does not insure that all aneurysms will be visualized. This is understandable on the basis of the dynamics of fluid flow in tortuous tubes, the size of some of the lesions and on the likelihood of partial thrombosis in some of the aneurysms. It is likely that repetition of the angiograms after an interval of time would visualize aneurysms in some cases.

In this series, aneurysms of the anterior communicating artery were the ones most frequently unvisualized by angiography.

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THE EFFECT OF CONTRAST MEDIA ON THE BLOOD-BRAIN-BARRIER*

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THE widespread popularity of the technique of cerebral angiography as a method for more precise diagnosis in selected cases has pointed up the fact that this method is not without morbid and even fatal sequelae. These occur even though errors of judgment in selecting patients and errors of technique and ineptitude in its employment are excluded.

It has been our experience and that of others[6] that these complications cover a wide range of possibilities from petechial hemorrhages of the periorbit and retina to loss of consciousness and death, including between these two extremes, severe burning paresthesias of the contralateral extremities, fine generalized muscular twitchings, convulsions, coma, and irregularity of cardiac and respiratory rate and rhythm.

FIG. 1. Flat plate of abdomen taken 2 years following internal carotid angiography using 36 cc. of colloidal thorium dioxide showing retention of medium in liver.

The incidence of such pathophysiologic complications occurs with great enough frequency to warrant search for cause, and development of methods for prevention. Diodrast, although rapidly excreted and physiologically acceptable, cannot satisfactorily be administered without analgesia because of discomfort induced. Colloidal thorium dioxide on the other hand, while innocuous in the above sense and therefore easily administered under local anesthesia and sometimes productive of greater contrast on films, is stigmatized by its radio-activity and the fact that

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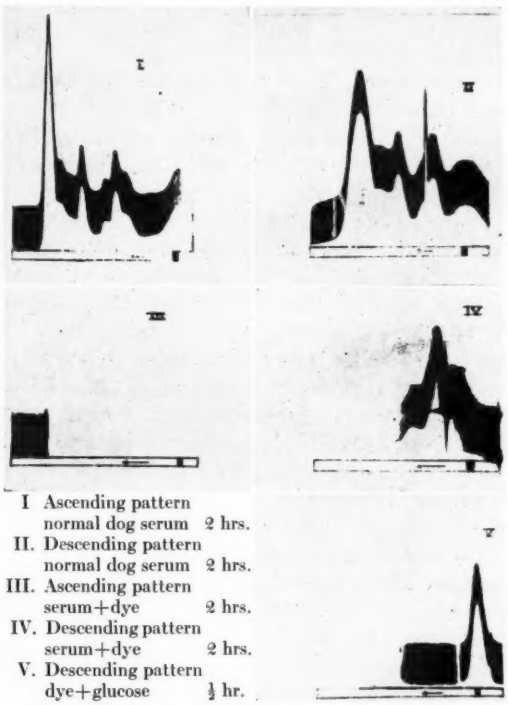
it is retained by the reticulo-endothelial system (Fig. 1).

We therefore attempted the search for a practical solution to the problem with materials now available, in the laboratory. All possible factors alleged to contribute to unwarranted side effects were analyzed.

METHODS AND MATERIALS

The basic method employed was that used by Broman and Olssen[1–5] in their earlier analysis of the problem. Using an acid aniline dye which is



I Ascending pattern normal dog serum 2 hrs.
II. Descending pattern normal dog serum 2 hrs.
III. Ascending pattern serum+dye 2 hrs.
IV. Descending pattern serum+dye 2 hrs.
V. Descending pattern dye+glucose ½ hr.

FIG. 2. Electrophoretic analysis. All run in phosphate buffer. 0.02 N. PO_4, 0.10 N. NaCl. pH 7.8 X = .0068 mhos. Current 15 ma. and 120 volts.

I and II are normal patterns for dog serum. III and IV are those obtained from serum of a dog given 50 mg./kg. of Evans blue. The plasma protein peaks are virtually obscured in these tracings by the presence of the opaque dye. V illustrates that the dye alone migrates at about 4 times the speed of the combined dye-serum.

taken up by plasma protein these authors demonstrated breakdown of the blood-brain-barrier (BBB). Dye takeup by the protein molecule is shown by electrophoretic analysis of plasma before and after injection of the dye (Fig. 2). The technique was modified by having the dye in circulation before injection rather than following injection of the contrast medium. Changes

incident to and associated with breakdown of the BBB were further examined by use of the Forbes' window for direct observation of pial vessels, by manometric and chemical study of the cerebrospinal fluid, and by histologic and clinical evaluation. Most of the experiments were acute.

The subjects used were 162 dogs and 12 rabbits.

Materials included in this analysis were unknown lots of 35 per cent diodrast, 70 per cent diodrast, 70 per cent Urokon, 35 per cent colloidal thorium dioxide, sodium iodide, and sodium chloride and glucose solutions of the same and greater tonicity as 35 per cent diodrast.

RESULTS

In general, we have been able to substantiate the work of Broman and Olssen[1–5] who have shown the basic lesion to be a disturbance of cerebral vascular permeability, and that concentration and time of exposure are of primary importance in determining the extent of this lesion.

Gross Vascular Changes. Neither acute spasm nor hemorrhage were observed. Arterioles varying in size from 80 to 90 microns were observed during injections of 35 per cent diodrast and did not change in diameter at any time more than 9 microns. Histologic changes in rabbits were found but were also seen in control animals in which simple ligation of the internal carotid artery had been carried out.

Given in doses considerably larger than those that one would use clinically, 35 per cent diodrast produced breakdown of the BBB in a percentage about equal to that of unfavorable sequelae after clinical angiography—that is, about 1 in 13 or 14 subjects (Table 1). Larger amounts of diodrast broke down the BBB with considerable regularity and caused convulsive seizures in 3 animals out of 26. In larger doses convulsive seizures were seen constantly (Table 2).

It was of interest to note that some protection was afforded by the presence of circulating dye. Those dogs that had dye injected either just preceding or following injection of the contrast medium recovered from fits, when they occurred, much more readily (Table 3). In some instances it was felt that even though breakdown was apparent and fits should have occurred the dogs were actually protected from convulsions by the presence of the circulating dye. The significance of this is a matter for speculation.

pH Variability. pH was excluded as a factor of possible significance in the effect on capillary permeability since random lots of all media tested were found to lie within the range pH 7.2 to 7.6.

Osmotic Pressure. Thirty-five per cent diodrast has a tonicity of approximately $3\frac{1}{2}$ times that of plasma. Solutions of sodium chloride and glucose of this approximate osmotic value were found generally to have a less pronounced effect upon cerebral capillary permeability, although in some instances the effect was equal to that produced by 35 per cent diodrast. A more hypertonic solution of glucose (4.5 × plasma tonicity) produced marked bluing in 1 case in which it was used (Table 4).

Air Embolism. In order to bring about breakdown of the cerebral capil-

TABLE 1

Effective dosage by clinical method

Exp. No.	Lot No.*	Amount Injected (cc.)	Time of Injection (sec.)	Rate of Injection (cc./sec.)	cc./gmb./sec.†	Bluing‡		Convulsions
41	A	3	3	1	0.011	0	0	0
50	A	3	4	0.75	0.009	0	0	0
40	A	3	4.7	0.64	0.007	0	0	0
42	A	3	4.9	0.61	0.007	0	0	0
43	A	3	6.1	0.49	0.006	0	0	0
38	A	8.6	1.6	5.1	0.064	0	0	0
48	A	8	3	2.7	0.031	0	0	0
39	A	8.1	8.6	0.94	0.001	+++	++	0
45	A	10	1.7	5.9	0.069	0	0	0
47	A	10	7	1.4	0.015	0	0	0
46	A	10	7.2	1.4	0.018	0	0	0
44	A	10	10	1	0.012	0	0	0
49	A	10	11	0.91	0.012	0	0	0
16	B	30	9.2	3.3	0.053	++	+++	0
26	B	30	17	1.8	0.026	++	+++	0
34	A	55	44	1.25	0.015	+	++	0
36	A	45	31.5	1.4	0.018	++++	++	0
53	A	50	22	2.3	0.032	+++	++	0
68	A	55	30	1.8	0.019	0	0	0
79	A	50	35	1.4	0.018	++++	+++	0
80	A	50	35	1.4	0.016	++++	+++	0
23	B	47	21	2.2	0.028	0+	+	0
10	C	61	24.2	2.5	0.032	++	+++	0
11	C	77	25	3.1	0.032	0+	0+	0
24	C	50	24	2.1	0.030	++	+++	0
19	D	47	16.7	2.8		+++	++++	++++
89	D	50	58	0.86	0.011	+	++	0
90	D	50	34.5	1.45	0.017	+	+++	0

* Refers to 35% diodrast unless otherwise specified.

† cc. per gram of brain tissue per second.

‡ Left column = intensity of response; right column = distribution of response.

lary barrier it was necessary to administer greater than lethal doses of air intravenously (Table 4).

Anoxia. The effect of anoxia was studied in two ways: Two dogs were given Evans blue and taken to a simulated altitude of 30,000 to 36,000 feet in the high altitude chamber where death ensued after 20 and 40 minutes respectively. A third dog, treated with intravenous Evans blue, was subjected to intermittent tracheal clamping until heart action ceased. There was no evidence of breakdown of the BBB (Table 4).

Other Media. Twenty-five per cent sodium iodide, 70 per cent Urokon, and 70 per cent diodrast all produced massive breakdown of the BBB. The first two substances, in addition, when given in doses of approximately 50 cc., produced a marked hemorrhagic effect.

TABLE 2

Comparison of lots by MB method—cumulative effect

Exp. No.	Lot No.	Amount Injected (cc.)	Time of Injection (sec.)	Application Time* (sec.)	Rate of Injection (cc./sec.)	cc./gmb./sec.	Method	Bluing		Convulsions	Tap on Nose†
Comparison of Lots by MB Method											
103	A	30	9	11.2	3.3	0.041	MB	+++	++++	0	+++
104	A	50	9	10.9	5.6	0.07	MB	+++	++++	0	+++
105	A	30	9	11	3.3	0.048	MB	++++	+++	0	+++
106	A	60+30	7+2=9	8+3=11	10	0.116	MB	+++	++++	0	+++
107	A	90	9	11	10	0.135	MB	+++	++++	++++	++++
108	A	90	9	15	10	0.115	MB	++++	++++	++++	++++
132	E	50	9	25	5.6	0.072	MB	0+	++	0	++
133	E	68	9	10.6	7.6	0.088	MB	+++	++++	0	++
134	E	90	9	10.4	10	0.111	MB	+	++	++	+++
135	E	45	9	10.4	5	0.066	MB	+	++++	+	+++
136	E	90	9	10.2	10	0.106	MB	+	++++	+++	++
137	E	80	9	10.7	9	0.122	MB	++++	++	0	++
138	F	90	9	10.7	10	0.108	MB	+	+	0	+
139	F	40	9	10	4.4	0.054	MB	+	+	0	0
140	F	60	9	10.8	6.7	0.074	MB	++	++++	+	+++
141	F	75	9	10.5	8.3	0.091	MB	+	+	0	+
142	F	60	10	11.1	6	0.077	MB	+	++++	0	+
143	F	90 60	7.2, 4.0=11.2	7.5, 4.1=11.6	13.4	0.14	MB	+	++	+	++
144	G	115	9	11.1	12.8	0.175	MB	++++	++	0	+++
145	G	70	9	10.5	7.8	0.095	MB	+++	+++	0	++
146	H	90	9	10.7	10	0.108	MB	++	++++	++	+++
147	H	50	9	10.5	5.6	0.057	MB	++	++	0	++
148	I	50	9	16.5	5.6	0.054	MB	+++	++++	0	+++
149	I	70	9	14.5	7.8	0.082	MB	+++	++	0	+++
150	I	70	9	10.6	7.8	0.077	MB	+	+++	+	+++
151	I	50	9	10.2	5.6	0.075	MB	++	++	0	+
152	(NaCl)	120	10.4	12.4	11.5	0.131	MB	++	++	0	0
153	(NaCl)	100	9	10.2	11.1	0.173	MB	++	++	0	0
154	(NaCl)	60	9	10.5	6.7	0.080	MB	0	0	0	0+
155	(NaCl)	50	9	10.5	5.6	0.081	MB	0	0	0	0
156	(NaCl)	70	9	12	7.8	0.105	MB	0+	++	0	0
157	(NaCl)	40	9	10.8	4.4	0.072	MB	0	0	0	0
158	(NaCl)	60	9	10.5	6.7	0.072	MB	++	+	0	0
159	(NaCl)	70	9	10.3	7.8	0.095	MB	0	0	0	0
Cumulative Effect											
119	A	20, 30, 30	2.5, 2.0, 2.0	3.1, 2.3, 2.6	12.3	0.184	MB	++++	+++	0	++
120	A	90, 15, 15	9, 1.5, 4.0	0, 3.7, 5.0	8.3	0.097	MB	+++	++	++	++++

* Time that all blood in pial vessel under observation was completely replaced by injected medium

† Refers to twitching induced by light tap on nose.

* Time that all blood in pial vessel under observation was completely replaced by injected medium
† Refers to twitching induced by light tap on nose.

TABLE 3

Comparison of methods with controls—protection by various substances

Exp. No.	Lot No.†	Amount Injected (cc.)	Time of Injection (sec.)	Application Time (sec.)	Rate of Injection (cc./sec.)	cc./gmb./sec.	Method‡	Bluing		Tap on Nose	Convulsions
Comparison of Methods with Controls											
34	A	55	44		1.25	0.015	C	+	++		0
36	A	45	31.5		1.4	0.018	C	++++	++		0
53	A	50	22		2.3	0.032	C	+++	++		0
68	A	55	30		1.8	0.019	C	0	0		0
79	A	50	35		1.4	0.018	C	++++	+++		0
80	A	50	35		1.4	0.016	C	++++	+++		0
94	A	90	9	11.2	10	0.112	TB	0	0	+	0
97	A	90	5	6	18	0.22	TB	++++	++++	+++	++
98	A	90	9	10.8	10	0.145	TB	+++	++	+++	+++
99	A	60	9	14.5	6.7	0.094	TB	++++	+++	+++	++
100	A	60	9	11.5	6.7	0.088	TB	0+	+	+++	++
101	A	120	9	11.6	13.34		TB	0	0	+++	+++
102	A	90	9	11.5	10		TB	0+	+	+++	+++
103	A	30	9	11.2	3.3	0.041	MB	+++	++++	+++	0
104	A	50	9	10.9	5.6	0.070	MB	+++	++++	+++	0
105	A	30	9	11	3.3	0.049	MB	++++	+++	+++	0
106	A	60	7+2	8+3			MB	+++	++++	+++	0
107	A	90	9	11	10	0.135	MB	+++	++++	++++	++++
108	A	90	9	15	10	0.115	MB	++++	++++	++++	++++
152	(NaCl)*	120	10.4	12.4	11.5	0.131	MB	++	++	0	0
153	(NaCl)*	100	9	10.2	11.1	0.173	MB	++	++	0	0
154	(NaCl)	60	9	10.5	6.7	0.080	MB	0	0	0+	0
155	(NaCl)	50	9	10.5	5.6	0.081	MB	0	0	0	0
156	(NaCl)	70	9	12	7.8	0.105	MB	0+	++	0	0
157	(NaCl)	40	9	10.8	4.4	0.072	MB	0	0	0	0
158	(NaCl)*	60	9	10.5	6.7	0.072	MB	++	+	0	0
159	(NaCl)*	70	9	10.3	7.8	0.095	MB	0	0	0	0
Protection by Various Substances										**Substance used to protect:**	
61	A	35	24		1.45	0.020	C	0+	0+	20 mg. Benadryl	0
67	A	55	32		1.72	0.020	C	+	++	10 mg. Benadryl	0
117	A	85	9	11	9.4	0.12	TB	0+	++++	200 mg. Rutin	0
118	A	50	9	9	5.6	0.077	TB	+	++	200 mg. Rutin	0
115	A	90	9	12.5	10.0	0.131	MB	0	0	200 mg. Rutin	0
116	A	50	9	10.5	5.6	0.069	MB	0+	+	200 mg. Rutin	0

† (NaCl) = 0.9% saline (* = injection made with all possible force).
‡ C = Clinical. TB = True Broman. MB = Modified Broman (i.e. indicator dye given *before* carotid injection).

TABLE 4

Exp. No.	Substance Injected	Amount Injected (cc.)	Time of Injection (sec.)	Application Time (sec.)	Rate of Injection (cc./sec.)	cc./gmb./sec.	Method	Bluing*		Convulsions	Histologic Lesion	Neurologic Change
Effect of Equal Osmotic Solution (Isotonic with 35% Diodrast)												
109	3.2% (NaCl)	90	18	18.5	5	0.057	MB	0+	+++	0		
110	3.2% (NaCl)	100	9	26	11.1	0.146	MB	0	0	0		
111	3.2% (NaCl)	100	9	12.2	11.1	0.13	MB	0+	++	0		
112	3.2% (NaCl)	70	9	12.5	7.8	0.096	MB	+++	++	0		
113	19.4% Glucose	100	8	10.5	12.5	0.157	MB	0	0	0		
66	3.2% (NaCl)	50	21.5		2.3	0.025	C	0	0	0		
88	19.4% Glucose	50	40		1.25	0.013	C	0	0	0		
87	19.4% Glucose	50	30.5		1.64	0.017	C	0	0	0		
72	20.3% Glucose	40	20		2	0.023	C	0	0	0		
65	25.0% Glucose	50	26.5		1.89	0.028	C	++++	++	0		
Effect of Anoxia												
57	30,000 ft. for 35 min., 36,000 ft. 5 min.→Death							0	0	0		
69	30,000–32,000 ft. for 18 min.→Death							0	0	0		
58	Trachea clamped 4×(1½–4 min.) over 45 min. period→Death							0	0	0		
Air Embolism												
54	2% NaCl:Air	50	25.5		1.96	0.024	C	+	++			Died
55	40 NaCl:10 Air	50	19		2.6	0.033	C	0+	++			Died
59	Air	5	4		1.25	0.014	C	0	0			Lived
60	Air	10	5		2	0.022	C	0	0			Died
Other Contrast Substance												
22	Thorotrast	45	17.7		2.54	0.036	C	0	0	0		
17	Thorotrast	43	19		2.26	0.032	C	0	0	0		
18	Thorotrast	48	20		2.4	0.032	C	0	0	0		
21	Thorotrast	44	20.6		2.14	0.029	C	0+	+++	0		
15	Thorotrast	68	27.8		2.44	0.037	C	+	+++	0		
78	70% Diodrast	48	50		0.96		C	++++	+++	0		
77	70% Urokon	12	29		0.41	0.005	C	+++ +H	++++	++++		
76	70% Urokon	48	40.7		1.18	0.017	C	+H	++++	++++		
82	25% NaI	13	15		0.87		C	++H	++++	+++		
81	25% NaI	48	45		1.07		C	0+H	++++	++++		
Dog Histology (Chronic Experiments)												
63	A	50	28		1.79		C			0	0	0
64	A	40	58		0.69		C			0	0	0
75	A	50	32		1.56		C			0	0	0
74	A	50	58		0.86		C			0	0	0
73	A	50	32		1.56		C			0	0	0
83	A	55	32		1.72		C			0	0	0
84	A	50	32		1.56		C			0	0	0

* H = Hemorrhagic lesion.

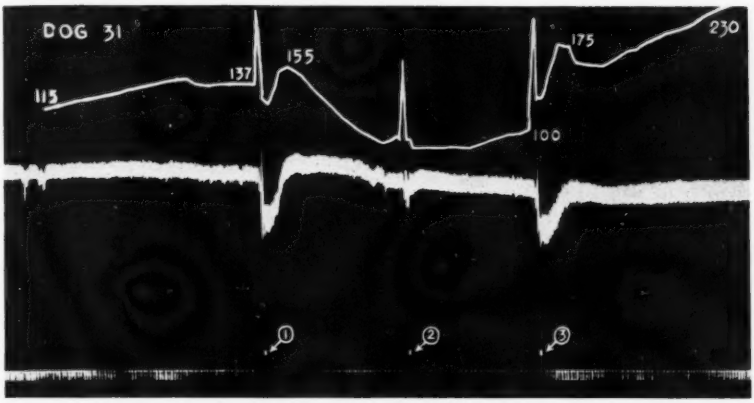


FIG. 3. Simultaneous measurement of CSF pressure (upper curve mm. of H_2O) and femoral artery pressure (lower curve) demonstrating post-injection sustained elevation of CSF pressure (3) unrelated to blood pressure values. (1) 60 cc. 35% diodrast. (2) 60 cc. 12.5% glucose. (3) 60 cc. 35% diodrast.

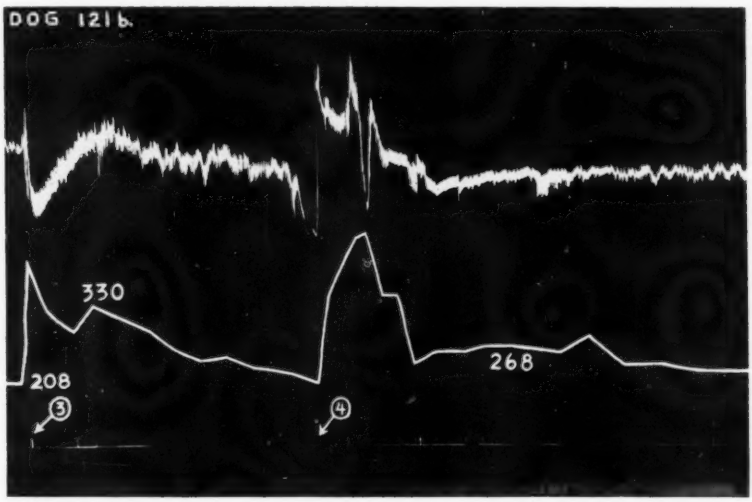


FIG. 4. Simultaneous measurement of CSF pressure (lower curve mm. H_2O) and femoral artery pressure (upper curve) showing slow recovery of CSF pressure increase following injection of 90 cc. of 35% diodrast (3) and parallel rise and fall of CSF and blood pressures associated with breathing 100% CO_2 for 2 minutes.

Changes in Cerebrospinal Fluid Pressure. Manometric studies demonstrated a post-injection rise in cerebrospinal fluid pressure not explainable in terms of blood pressure changes in 4 out of 5 dogs injected with 35 per cent diodrast. No rise was observed with hypertonic saline or glucose controls. On the other hand, the rise in cerebrospinal fluid pressure following

carbon dioxide inhalation was transient and paralleled blood pressure rise (Figs. 3 and 4).

Changes in Cerebrospinal Fluid Protein. Presumably, protein could enter the cerebrospinal fluid as it crosses the Virchow-Robin spaces in passing from blood to brain. It is difficult to conceive of any source other than a blood-vascular one to account for sudden rise in cerebrospinal fluid protein following carotid injection. In two experiments the cerebrospinal fluid protein demonstrated an increase approximating 40 per cent following injection of 35 per cent diodrast (Table 5).

TABLE 5

Effect on CSF protein

Exp. No.	Lot No.	Amount Injected (cc.)	Time of Injection (sec.)	Method	CSF Protein Before Injection	CSF Protein ½ Hr. After Injection	Percentage Change	cc./gmb./sec.	Convulsion
27	11.6%, Glucose	50	28	C	32	28	−12%	Control	0
28	11.6%, Glucose	70	31	C	28	27	−3.6%	Control	0
35	12.5%, Glucose	100	80	C	16	22	+37.6%	Control	0
51	A	60	24	C	18	17	−5.6	Diodrast	0
52	A	30	21.5	C	25	35	+40	Diodrast	0
122	A	90	9	TB	12	97	+800	Diodrast	++++
123	F	90	10	TB	14	53	+280	Diodrast	++++
127	NaCl	100	9	TB	19	22	+15.7	Control	0
128	NaCl	100	9	TB	12	20	+66	Control	0
130	108DLO2	90	9	TB	43	198	+360	Diodrast	0

SUMMARY

From the above evidence it would appear that the fundamental lesion of the cerebral circulation is one of alteration of the endothelial capillary membrane or the BBB as demonstrated by Broman and Olssen.[1–5] Furthermore, the extent of such a lesion is directly proportional to the *concentration* and *quantity* of test substance in contact with the capillary endothelium and the *time* during which this concentration prevails. Repeated fractional doses have a cumulative effect.

Other factors such as pH of available preparations, air embolism, vasospasm and anoxia have no part in initiating this breakdown.

Breakdown of the BBB is accompanied by passage of protein-rich fluid into the extravascular compartment of the brain, and by a rise in cerebrospinal fluid pressure. It can therefore be postulated that in human subjects with intrinsic vascular disease, subsequent spasm, thrombosis, and thromboembolism are probably secondary to the initial alteration of permeability.

It is possible that some protection from this pathological change can be introduced; however, from a more practical point of view for the present, greater care in selection of patients who might conceivably have *intrinsic arterial disease* and the use of minimally effective concentrations of contrast media over a shorter period of time are more practical methods of avoiding these complications.

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THE CEREBRAL CIRCULATION IN POSTOPERATIVE INTRACRANIAL HYPOTENSION*

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THE syndrome of postoperative intracranial hypotension has been frequently described as a persistent semicomatose or lethargic state following intracranial surgery associated with a low cerebrospinal fluid pressure. It is most often seen after evacuation of a chronic subdural hematoma and following the relief of an obstruction causing a chronic internal hydrocephalus. It has been assumed that the decrease in cerebrospinal fluid pressure was causally related to the protracted disturbance of consciousness. Intrathecal instillation of fluid to restore cerebrospinal fluid pressure has been suggested frequently as treatment for this syndrome.

Chorobski[1] has recently reviewed the theories of the pathogenesis of postoperative intracranial hypotension and agreed with the suggestions that this syndrome was caused by cessation or insufficient formation of cerebrospinal fluid.

Leriche[3] first suggested that the clinical picture was the result of cessation of production of cerebrospinal fluid caused by cerebral vasoconstriction and ischemia of the choroid plexus. Wolff[5] agreed that a reduced formation of cerebrospinal fluid is at fault and ascribed this to a disturbed venous circulation of the choroid plexus.

Chorobski[1] postulated that as a result of the operative procedure there occurs a decrease in cerebral blood flow causing an ischemia of the diencephalon. He further assumed that a center exists in the latter structure controlling production of cerebrospinal fluid from the choroid plexus together with a center for consciousness. The disturbance of these centers by the ischemia was suggested as the origin of the syndrome of postoperative intracranial hypotension. It thus appears that it would be of interest to report on the state of the cerebral circulation in this syndrome now that a reasonably accurate method for its determination in man is available.

MATERIAL AND METHOD

Three patients were studied within 3 to 5 days after evacuation of typical chronic subdural hematomas. All 3 patients failed to respond in the usual manner following surgery, remaining semicomatose or comatose, and a postoperative lumbar puncture in each instance revealed a profound cerebral hypotension. The first 2 patients, both of whom remained comatose following evacuation of bilateral subdural hematomas, were treated with

* Aided by a grant from the U. S. Public Health Service.

normal saline instillation into the lumbar subarachnoid sac. The first patient, aged 45, survived, requiring 4 weeks for consciousness to be fully regained. The second patient, aged 65, died 1 week after operation and 3 days after the study reported in this article had been performed. The third patient made a prompt recovery after institution of therapy consisting of carbon dioxide inhalations and intravenous fluids. At no time was the condition of this last patient as serious as the others.

The nitrous oxide technique as originally described[2] was used. Mean arterial blood pressure was recorded directly from the femoral artery by means of a damped mercury manometer. Cerebrospinal fluid pressure in the lumbar subarachnoid sac was recorded continuously upon a water manometer with the patient lying supine upon a mattress originally designed for use with patients receiving continuous spinal analgesia. It has been observed that the spinal fluid pressure is consistently at least 50 to 75 mm. of H_2O higher in the supine position than in the lateral recumbent position.

RESULTS

Reference to Tables 1 and 2 shows that in all patients studied a diminished cerebral blood flow was associated with the reduced cerebrospinal

TABLE 1

Blood gas constituents in patients with intracranial hypotension

Pt.	Age	Sex	C.S.F.P.		M.A.B.P.		Arterial								Internal Jugular							
							O_2 Content		CO_2 Content		CO_2 Tension		pH		O_2 Content		CO_2 Content		CO_2 Tension		pH	
			I	II	I	II	I	II	I	II	I	II	I	II	I	II	I	II	I	II	I	II
			mm. H_2O		mm. Hg		Vol. %		Vol. %		mm. Hg				Vol. %		Vol. %		mm. Hg			
1	46	M	30	174	94	94	11.7	11.3	52.5	51.1	35	34	7.48	7.48	6.5	6.0	57.4	56.1	40	40	7.44	7.43
2	65	M	22	190	78	83	15.1	15.0	45.9	44.6	34	33	7.46	7.46	8.9	7.7	51.2	52.1	38	38	7.42	7.42
3	38	M	38	106	81	85	19.4	19.9	40.8	43.6	38	43	7.36	7.33	12.4	14.7	47.7	48.6	40	47	7.33	7.30

C.S.F.P. Cerebrospinal fluid pressure.
M.A.B.P. Mean Arterial Blood Pressure.
I Initial Study.
II Study following intrathecal instillation of normal saline in patients 1 and 2, and 5% CO_2 inhalations in patient 3.

fluid pressure. The reduction in cerebral blood flow ranged from 30 to 46 per cent below the average normal as measured in healthy young males.[2] The cerebrovascular resistance in each patient was clearly elevated and correlated well with the reduction of cerebral blood flow. The mean arterial blood pressure was within normal limits in each patient. The oxygen consumption was generally reduced and correlated well with reduced state of consciousness observed at the time of the study.

In the first two patients following the initial observations, normal saline solution was inserted into the spinal subarachnoid space sufficient to raise the cerebrospinal fluid pressure to normal. This resulted in some, but not a

striking increase in cerebral blood flow. In any event the cerebrovascular resistance was not materially altered by this procedure. In the second patient a marked increase in cerebral oxygen uptake was observed but this was not accompanied by any evidence, clinically, of improvement in the patient's state of consciousness.

The third patient was given 5 per cent carbon dioxide by inhalation and the cerebral circulation was redetermined. The cerebrovascular resistance was reduced to a normal level and the cerebral blood flow restored to a normal level. The mean arterial pressure increased somewhat but this change was far less striking than the reduction of cerebrovascular resistance. The carbon dioxide inhalations increased the cerebrospinal fluid pressure from 40 to 100 mm. of water. Cerebral oxygen consumption was unaffected.

TABLE 2

Cerebral circulatory functions in patients with intracranial hypotension

Pt.	A-V.O_2		R. Q.		C.B.F.		C.M.R.O_2		C.V.R.	
	I	II	I	II	I	II	I	II	I	II
	vol. %				cc./100 gm./min.		cc./100 gm./min.		mm. Hg / cc./100 gm./min.	
1	5.2	5.2	.94	.96	38	40	2.0	2.1	2.5	2.7
2	6.2	7.3	.86	1.03	29	33	1.8	2.4	2.7	2.5
3	7.0	5.2	.99	.96	35	50	2.4	2.6	2.3	1.7

C.B.F.—Cerebral blood flow.
C.M.R.O_2—Cerebral oxygen consumption.
C.V.R.—Cerebrovascular resistance.
I & II as in Table I.

DISCUSSION

The reduction of cerebral blood flow observed in each instance in this study could hardly be the result of reduced cerebrospinal fluid pressure. The pressure of the cerebrospinal fluid is an expression of a factor offering resistance to the flow of blood in the cerebral vessels and a decrease in this pressure should tend to increase the cerebral blood flow. On the other hand it has recently been shown,[4] and we have confirmed to our satisfaction, that the rate of the cerebral circulation is an important factor in the maintenance of cerebrospinal fluid pressure. The correct interpretation undoubtedly then, is that the reduced cerebral blood flow in these patients contributes to the reduction of cerebrospinal fluid pressure, rather than vice versa.

The reduced cerebral blood flow was the result of the increased cerebrovascular resistance insofar as the mean arterial blood pressure was consistently within normal limits. The increased cerebrovascular resistance can only be interpreted as being caused by a decreased vascular diameter, probably the result of spasm, since there was no reason to believe that the vis-

cosity of the blood was increased and indeed all of the patients studied were somewhat anemic.

The origin of the cerebrovascular spasm in instances of postoperative or post-traumatic intracranial hypotension can only be a matter for conjecture. Nevertheless it seems reasonable to believe that the production of cerebrospinal fluid is curtailed by the reduced cerebral circulation and this further contributes to, if it is not the principal source of, the intracranial hypotension.

The reduced cerebral oxygen consumption correlates well with the depressed states of consciousness that these patients exhibited. That this was not immediately reversible is indicated by the fact that in the third patient, despite the prompt return of his cerebral circulation to virtually normal levels, the cerebral metabolic rate as measured by oxygen consumption was unaltered. It might be presumed that the prolonged disturbance of circulation so affected the enzyme systems of the brain that the metabolic rate was depressed and that these changes are not immediately reversible. That they are reversible was demonstrated by the eventual satisfactory recovery of two of the three patients studied.

The analysis of these data would suggest that reasonable therapy of the intracranial hypotensive syndrome would be directed towards the restoration of the cerebral circulation by relieving the cerebrovascular spasm. The most powerful selective cerebrovascular dilator available is carbon dioxide. In the single instance in this series in which it was tried, it did indeed prove effective and in addition it also tended to restore the cerebrospinal fluid pressure towards a normal level. To increase the cerebrospinal fluid pressure directly by intrathecal injection of fluid, while not proving deleterious in the instances in which its effects were measured, for the moment appears unphysiological.

SUMMARY

The cerebral circulation was studied in three patients suffering with intracranial hypotension following the evacuation of a subdural hematoma. The cerebral blood flow was found to be markedly reduced in each instance as the result of an increased cerebrovascular resistance. It is postulated that the increased cerebrovascular resistance was caused by cerebrovascular spasm and that reasonable therapy of the intracranial hypotensive syndrome would consist of attempting to restore the cerebral circulation to normal by relieving the cerebrovascular spasm.

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PERSONAL EXPERIENCE IN THE SURGICAL TREATMENT OF EPILEPSY*

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One of the most impressive and dramatic symptoms of disease of the nervous system is an epileptic attack. The neurosurgeon sees a good number of patients afflicted with various types of epilepsy. Sometimes this symptom is only part of a clinical picture obviously produced by an intracranial lesion, like tumour, scar or atrophy. But in other instances the evidence of a cerebral lesion is not so clear, in spite of the great help afforded by radiographic and electroencephalographic auxilliary methods. Although all neurosurgeons are agreed upon the necessity of operation in cases of epilepsy secondary to definite cerebral lesions, there are some differences of opinion concerning those cases in which the criteria under consideration are mainly functional.

Our personal tendency in the surgical management of epilepsy is towards a careful selection of patients, and radical excision of the affected epileptogenic regions of the brain, whenever this is possible, while taking care to avoid causing great neurological disability. Few epileptic patients should be operated on, but with those selected for this type of treatment, the neurosurgeon, with a knowledge of the pathophysiology of the disturbance, must be radical in his efforts to remove the affected regions.

In reviewing our material of the last 5 years it was found that in a series of 755 major intracranial operations, there were 130 patients (17 per cent) with epilepsy. These cases may be divided into the following groups.

I. Expansive intracranial lesions (90 cases)
II. Focal cerebral lesions
  A. Localized cerebral scars (11 cases)
  B. Cerebral atrophies (19 cases)
III. Psychomotor and other functional disturbances (10 cases)

I. EXPANSIVE INTRACRANIAL LESIONS

Epileptic seizures were an important symptom in about a fourth of our patients with intracranial expansive lesions. The incidence was of course much higher among the patients with supratentorial lesions, nearly half of these having a history of attacks.

The distribution of the cases according to the pathological type of the lesion was as follows:

* Presented at the VIII International Congress of Surgery organized by the Spanish Chapter of the International College of Surgeons, Madrid, May, 1952.

Gliomas	49
Meningiomas	14
Tuberculomas	5
Metastatic tumours	5
Hemangiomas and angiomas	4
Hematomas (intracerebral and subdural)	3
Abscesses	3
Cysticercosis	2
Hydatid cysts	2
Third ventricle tumours	3

In the group of supratentorial gliomas, epileptic manifestations were present in 35 per cent of the patients. As is well known, with some of the slower growing gliomas (oligodendrogliomas, astrocytomas) epileptiform symptoms may be present for many years. There is a wide variation in the type of the attacks, depending on the localization of the tumour.

With supratentorial meningiomas the incidence of epilepsy was similar (3[illegible] per cent). It was the predominant symptom in cases of meningioma of the convexity and parasagittal region near the Rolandic areas. Jacksonian motor attacks were the most frequently recorded. The duration of the epileptiform symptoms varied from 1 to 15 years. Another important practical point is that definite signs of increased intracranial pressure were absent in many of these patients.

Tuberculomas represent about 7 per cent of our total series of 370 intracranial tumours. Although more frequently localized in the posterior fossa, those of the cerebral hemispheres often give rise to Jacksonian focal attacks because of their situation near the Rolandic areas.

No particular comment need be made on the rest of these lesions. However, an interesting feature of our material is the number of parasitic cysts. Cysticercosis cysts represent 3 per cent of our series, but produced epileptic symptoms in only a few cases; signs of increased intracranial pressure were more frequent. Hydatid cysts represent 2 per cent, and were usually localized in the cerebral hemispheres (7 of 8 cases). They sometimes caused epileptiform manifestations.

In these cases of epilepsy caused by expanding intracranial lesions, the neurosurgeon makes an effort to extirpate the abnormal mass and to leave a cerebral scar as clean and as small as possible, removing all the damaged cortex. The results of such operations are usually evaluated on the basis of the late survival period and not by the presence or absence of postoperative attacks. Definite figures cannot be given on the results in our material, which is presented mainly to show the variety of pathological lesions. However, from different sources it appears that at least half or more of the survivors have postoperative epilepsy. As shown by Penfield and Erickson,[13] the incidence of postoperative convulsions is higher with gliomas (astrocytomas) than with meningiomas.

II. FOCAL CEREBRAL LESIONS

A. *Localized Cerebral Scars.* In our series there are 11 cases of localized cerebral scars caused by trauma, bullets or circumscribed infections: 5

were in the frontal poles, 3 in the Rolandic region, 2 in the parietal lobe and 1 was in the occipital pole. Most of the patients were young; only 3 were over 30 years of age. In 6 cases the cerebral lesion was produced by a head injury and in 5 the causes were gunshot wounds. A history of infection of the primary wound with local abscess formation was obtained in 3 instances.

An interesting feature of post-traumatic epilepsy is the interval between the time of injury and occurrence of attacks. Many patients, in spite of a very severe post-traumatic scar in the brain, do not have epileptiform attacks until years after the injury. The electroencephalographic technique has been of help in some of these cases by showing abnormal discharges in the absence of clinical symptoms, and in such instances the cerebral scar may be removed at the time of the usual bone repair.

In our series of epileptics the intervals between the time of the injury and the appearance of the first attack varied from a few months to 11 years. In only 3 cases was the interval short (3, 9 and 15 months). In the rest it was long: in 5 cases between 5 and 6 years after injury, in 1 case after 8 years, and in 2 cases after 10 years. These figures confirm the view that the development of epilepsy after injury to the brain is slow and progressive. Sometimes early fits may occur within the first weeks after the injury, disappear completely for several years and then reappear to persist in the usual recurrent manner. This happened in one of our cases. These examples show the distinction between the immediate and late forms of post-traumatic epilepsy. They seem to have a different pathogenesis.

In the majority of our post-traumatic cases the attacks were referred to in the history as generalized seizures although sometimes a focal component or adversive beginning was noticed. With scars in the Rolandic areas there were typical Jacksonian fits which often progressed into a generalized discharge. Psychomotor automatic states were recorded in some cases of frontal scars.

An electroencephalographic study was performed in 8 cases and, with one exception, abnormal discharges were recorded in the injured area, such as the typical and sporadic spikes and sharp waves together with slow waves (4 cases) or only slow high voltage waves (3 cases). In one of the latter the slow waves of the occipital region spread to the other occipital lobe.

In all cases we performed an excision of the meningocerebral scar. The excisions in the frontal or occipital poles were more extensive than in the Rolandic areas. Sometimes we tried the effects of electrical stimulation of the atrophic convolutions at the border of the cerebral scar in an effort to determine the firing epileptogenic points. In the latter cases electrocorticography was also carried out and found useful in showing the region where spontaneous or provoked fits were initiated. When there was enough evidence the abnormal epileptogenic parts of the cortex were included in the surgical resection, but often the results of the electrical stimulation were disappointing and either no responses could be obtained or a generalized fit spoiled the examination.

In resecting the cerebral scars the white matter often appeared very hard and in those cases the excision was extended to the ventricular wall, especially when the scars were in the frontal and occipital regions. In two patients a bullet was still lodged in the brain, and after resecting the meningocerebral scar at the point of entrance, the bullet was also removed. In one case it lay against the falx and over the corpus callosum, and in the other it was on the floor of the anterior fossa. After resections of scars the

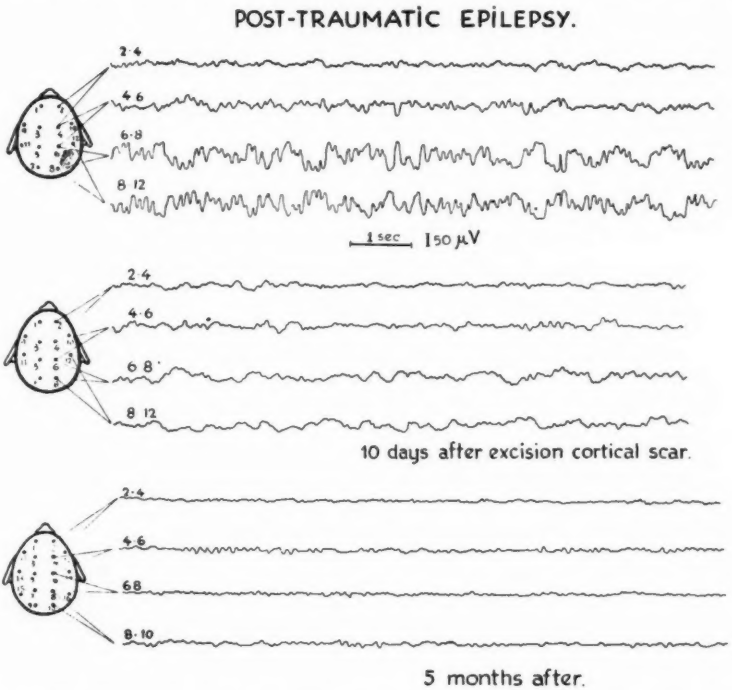

FIG. 1. Electroencephalograms of an epileptic who had had an injury of the right occipital lobe. Note improvement of the records after removal of the cortical scar.

defect in the dura was repaired with temporal fascia or with polyethylene film, and in some cases acrylic plates were used for bone defects.

It is extremely difficult to evaluate the results of operations performed for post-traumatic epilepsy. It is obvious from the histories of these patients that convulsions may develop many years after the injury. Therefore the postoperative course must be followed for a very long period before a real evaluation may be made. All that can be done for these patients is to remove a very sclerotic meningocerebral scar and leave another clean one with better preserved and vascularized edges. In some cases the electroencephalogram may indicate a favorable prognosis by showing normalization of the

electrical record after surgical removal of the scar and surrounding convolutions (Figs. 1 and 2). Another factor that complicates the assessment of the surgical results is the effect of anticonvulsive drugs which most patients take for some time after the operation.

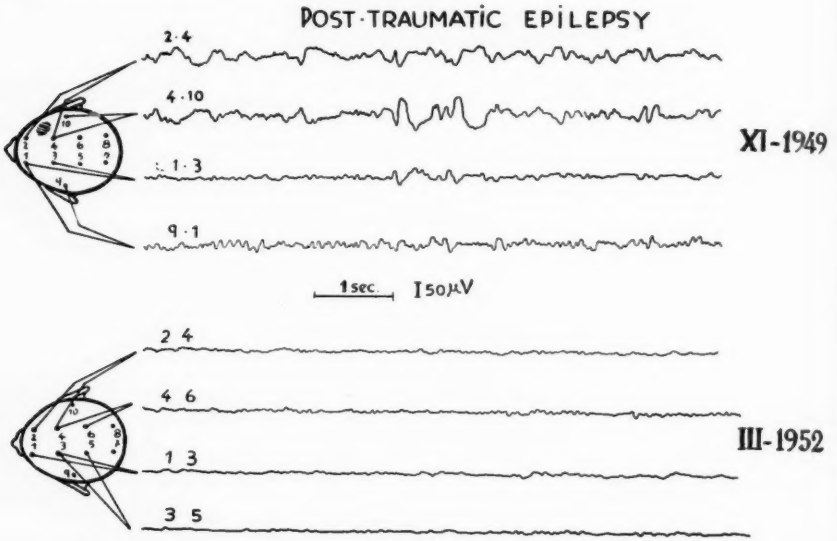

FIG. 2. Pre- and postoperative electroencephalograms in a case of post-traumatic epilepsy produced by a meningocerebral scar of the right frontal lobe. More than 2 years after frontal lobectomy, the attacks and also the abnormal waves have disappeared.

A summary of the results in our small series, based on reports obtained from 1 to 4 years after operation, is given in Table 1.

It appears from Table 1 that better results are obtained in those cases in which the scars are localized in the frontal or occipital poles, where one can make a wider excision of the surrounding tissue in the way of a partial

TABLE 1

Results

	Unknown	Bad (Fits Continue)	Improvement (Fits Diminished)	Good (Without Fits)
5 frontal scars and 1 occipital scar	1	—	1	4
3 scars of Rolandic region (all patients had increased motor deficit after operation)	1	1	—	1
2 scars caused by bullets, with a long intracerebral lesion. Removal of scar of entrance and bullet	—	2	—	—

or total lobectomy. Our small figures agree with those of Penfield and his coworkers[13,14] and Walker.[17]

B. *Cerebral Atrophies.* There were 19 patients with atrophic lesions of the brain producing epileptic seizures who were treated surgically. The lesions may be subdivided into the following groups: (a) cerebral hemiatrophy, (b) porencephalic cysts and malformations and (c) cortical atrophy of unknown origin.

(a) The group of cerebral hemiatrophies is the largest and most interesting one. There were 12 patients; most of these were children or young persons between 5 and 18 years of age; 2 were men, 27 and 30 years old respectively. The cause of the disease was apparently known in only half of the cases and was recorded as anoxia and trauma at birth (3 cases), meningitis (2 cases) and an infectious fever disease (1 case).

The epileptic manifestations started usually in early childhood and always before the age of 10 years. Sometimes the first attack was followed by a hemiplegia and in all cases examination revealed a typical picture of infantile hemiplegia with a definite mental defect and deterioration. The epileptic fits were usually focal Jacksonian in character; after local initiation a generalized spread of the seizure often developed. In some patients there were also associated generalized convulsions, petit mal attacks and psychomotor states. The epileptic symptoms recurred frequently in all cases.

Radiographic studies of these patients with infantile hemiplegia conform to the well-known picture of cranial and cerebral hemiatrophy, with different degrees of enlargement of the homolateral ventricle. The electroencephalographic disturbance was also very marked and abnormal discharges were always recorded from the affected hemisphere. Foci of spikes and slow waves were quite frequent over the side of the lesion, but in most cases the electrical disturbance was more diffuse and bilateral, although the abnormality dominated in the diseased hemisphere. A picture of fast spiky activity on the side of the lesion together with slow waves on the other side was also obtained. Besides the focal and propagated disturbances we observed in some records the presence of synchronous and bilateral slow discharges.

The operative procedure was completely different in the first and second half of these 12 cases of infantile hemiplegia with epilepsy. In the first 6 cases only a limited removal of the cystic atrophic areas or partial resection of the most affected cortical regions was performed. Electrical stimulation for detecting the most irritable points of the exposed cortex was used in some of the cases. The results as a whole were disappointing and the seizures usually persisted, although sometimes an improvement was noticed. One of the patients has been free of attacks for more than $1\frac{1}{2}$ years since a partial excision, although an electroencephalographic focus persists. In another of these patients there was a striking diminution in frequency of the fits together with an impressive change in the electroencephalogram 2 years after a limited cortical removal of a very hard and extensive lesion of the left cerebral hemisphere (Fig. 3).

These generally unsatisfactory results after limited surgical resections led to the trial of hemispherectomy or hemidecortication by removing all the cortex of the affected hemisphere and sparing the basal ganglia (Fig. 4). The first time we performed this operation was in 1949.[11] Up to the present we have done six hemispherectomies for this condition, some of which have been briefly reported.[10] Two patients died in the immediate postoperative period. The other 4 are doing well 3 years, 1½ years, 1 year, and 6 months after operation.

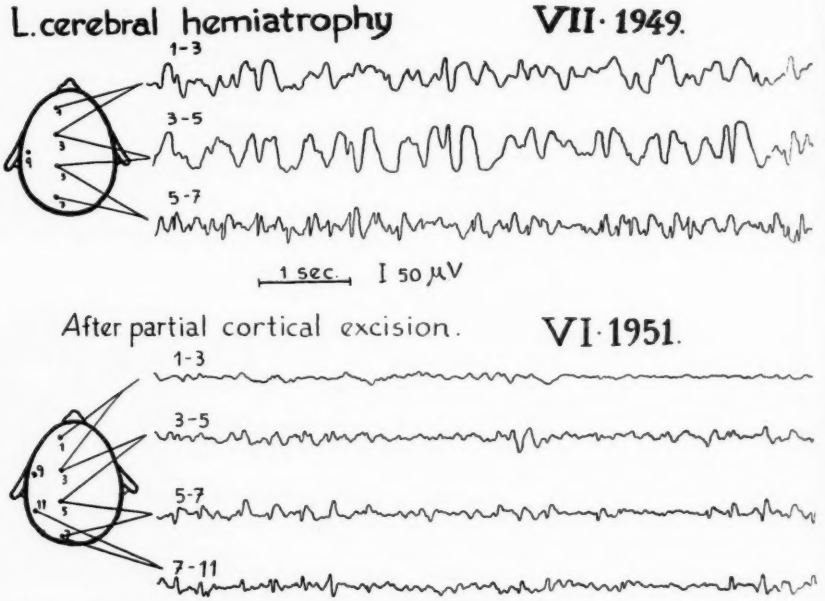

FIG. 3. A case of left cerebral hemiatrophy producing epilepsy. The electroencephalograms show improvement 2 years after a limited cortical excision of the affected hemisphere.

Our personal impression at the moment is that much better results are obtained by hemispherectomy than by partial resections, but hemispherectomy is also more dangerous, and may not solve the whole problem of epilepsy in these cases. Our first patient is free of attacks, with a complete absence of abnormal discharges in the electroencephalogram 3 years after the hemispherectomy. But in the other 3 cases there are still some abnormalities in the electrical recording from the remaining hemisphere (Figs. 5 and 6). Although all these 3 patients have improved tremendously after the operation and the fits have decreased markedly in frequency, some slight epileptiform manifestations still appear in 2 of them and even more rarely in the third (4 slight attacks in 1 year).

There is no doubt that hemispherectomy is a good operation for the treatment of severe epileptic disturbances of the infantile hemiplegias

(Krynauw,[7] Cairns and Davidson[2]) but it must be emphasized that besides its definite surgical risk there is the possibility that some electrical abnormalities and infrequent clinical discharges may persist after the operation. The subcortical origin of the seizure discharges has been substantiated by recent

FIG. 4. Surgical specimen in 1 of our 6 cases of hemispherectomy or hemidecortication. These patients had marked hemiatrophy causing infantile hemiplegia and epilepsy.

studies of deep electrical recording in epileptics (Hayne, Belinson and Gibbs,[6] Meyers *et al.*,[9] and Spiegel and Wycis[15]). There is also the possibility that there are abnormal areas in the opposite hemisphere or that the long-lasting abnormal discharges of the patients with cortical hemiatrophy have induced functional changes in the other hemisphere and perhaps these may subside in the course of time after the removal of the affected hemisphere.

(b) There were 3 patients with porencephalic cysts and malformations. One was a child with absence of the corpus callosum associated with cerebral

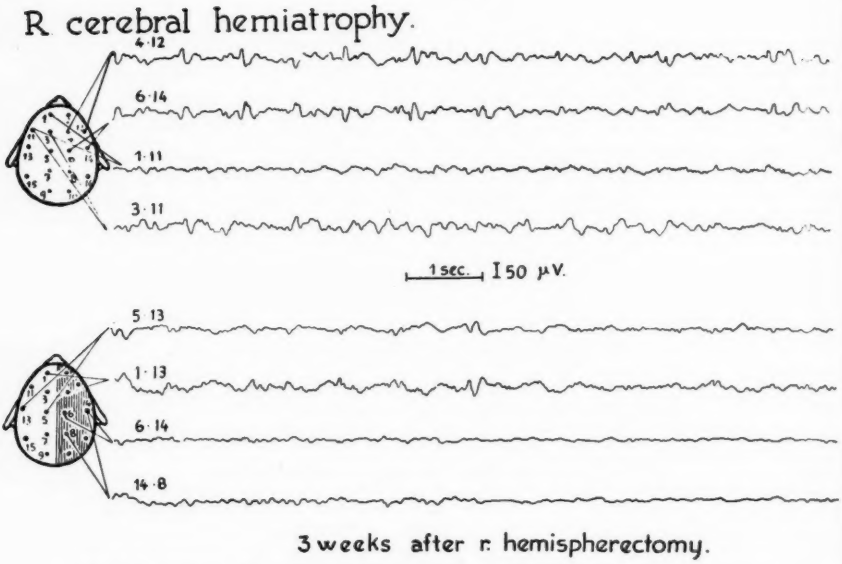

FIG. 5. Electroencephalograms before and 3 weeks after hemispherectomy in a case of right cerebral hemiatrophy.

malformation. Two were young men (21 and 26 years old) who began to have fits at the age of 18, caused by large cerebral cysts. In one an absent temporal lobe was replaced by an enormous cyst isolated from the ventricle by a fine membrane. In the other there was also a large paraventricular cyst, in the parietal region. All that could be done with these 2 patients was to open the cysts widely and form a communication with the ventricular system, but the attacks persisted. Drew and Grant[3] reported a series of porencephalic cysts with postoperative improvement in the seizures in a large proportion of the cases. They presumed that the favourable results

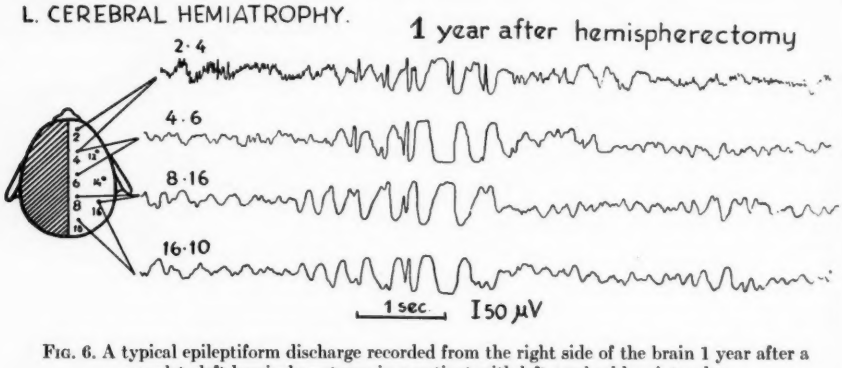

FIG. 6. A typical epileptiform discharge recorded from the right side of the brain 1 year after a complete left hemispherectomy in a patient with left cerebral hemiatrophy.

might be attributable to relief of tension by removal or opening of the cyst.

(c) Finally there were 4 patients (aged 18, 30, 46 and 52 years) with focal epileptiform attacks. Neurological examination disclosed some positive findings (monoparesis, some aphasia, sensory disturbances) in 2 of these cases; in the other 2 the findings were negative. The electroencephalograms showed some focal disturbances. Although the operations were performed with strong suspicion of a cerebral neoplasm, only some cortical atrophy of unknown origin was found. In 3 patients nothing more than a surgical exploration was done and the epileptic manifestations have continued. In the last case a cortical resection of the most atrophic parietal region was carried out and the fits have disappeared in the 3 years since the operation was performed.

III. PSYCHOMOTOR AND OTHER FUNCTIONAL DISTURBANCES

This last group of epileptic patients is even more complex because the localization and treatment of the epileptiform area are based on functional evidence only. In fact one can affirm that this section of neurosurgical treatment has emerged from the recent development of electroencephalographic techniques. There are 10 examples in our series: 8 patients with psychomotor epilepsy treated by anterior temporal lobectomy and 2 patients on whom we tried the effect of stereotaxic lesions of the thalamus. The histories of the first 7 patients with psychomotor epilepsy have already been reported[8] and the results are in agreement with the larger experience of Bailey and Gibbs[1] and Green and coworkers.[5]

The patients with psychomotor epilepsy were young, from 22 to 38 years old. While 4 presented a history of pure psychomotor seizures, the other 4 had in addition some generalized convulsive attacks. In the purer psychomotor forms the electroencephalographic studies demonstrated the typical abnormal discharges in the temporal regions with spikes and sharp waves and sometimes also some slow waves, as described by Gibbs, Gibbs and Fuster.[4] In the other 4 patients, with a combination of psychomotor and generalized seizures, besides the focal temporal discharges other more diffuse dysrhythmic abnormalities were found.

The operation performed in all these cases was removal of the more electrically abnormal temporal lobe. The excision corresponded to the tip of the temporal lobe, and was about 3 to 5 cm. in length. All this anterior temporal tissue was removed by suction. Before lobectomy, electrical stimulation under local anaesthesia and electrocorticography were carried out. There were no marked neurological or psychical changes following the anterior temporal lobectomy, which was performed on the left dominant side of the brain in 3 cases and on the other side in 5.

The results have been followed for only $1\frac{1}{2}$ to nearly 3 years after operation. They have been generally good in the group of patients who had psychomotor seizures only; the attacks have not recurred in the period of observation ($1\frac{1}{2}$ to 2 years) and only 2 of these patients have experienced occasional slight visceral sensations without any spread or form of seizure.

There has also been a great improvement in the electroencephalograms, and the spikes and abnormal temporal discharges have disappeared or greatly diminished (Fig. 7). An interesting fact that was noted in the serial electroencephalographic studies in our cases is the progressive normalization of the record in the months and years after temporal lobectomy, and this change had no relation with any medical anticonvulsive therapy.

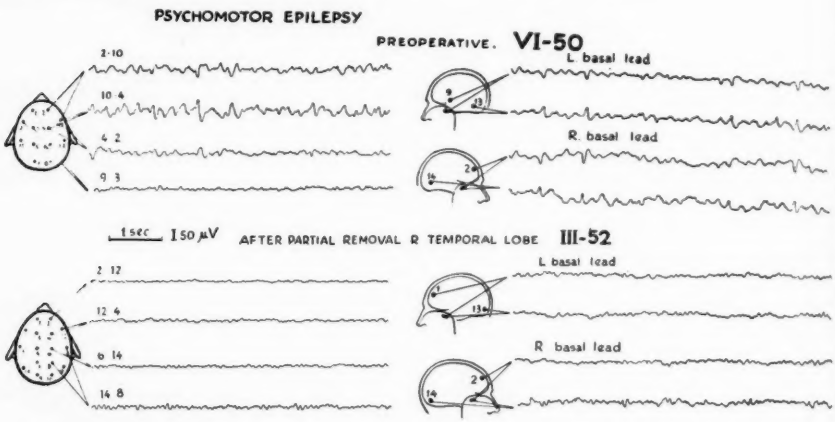

FIG. 7. An example of psychomotor epilepsy treated by right temporal lobectomy, showing the great improvement in the electroencephalogram nearly 2 years after operation. This was accompanied by a good clinical result.

In contrast with this favourable response in the group of pure psychomotor seizures, anterior temporal lobectomy was not very useful in those other 4 patients with a combination of psychomotor and generalized convulsive attacks. Although there was some occasional postoperative improvement in the electroencephalographic records, the seizures have persisted. It seems, therefore, that the surgical treatment for psychomotor epilepsy should be limited to the purer clinical forms, with a well-localized electrical focus in the anterior temporal region.

Finally in 2 epileptic patients we tried the therapeutic effects of making small stereotaxic lesions of the thalamus. One of them, with a curious localized myoclonic form of attacks, has already been reported,[12] and some improvement was obtained. In the experience of Spiegel, Wycis and Reyes[16] it is not possible to abolish completely the subcortical petit mal attacks by diencephalic stereotaxic lesions. They believe that there is a relatively wide area where the abnormal discharges may originate and therefore small lesions produce only partially successful therapeutic results. Further neurophysiological and neurosurgical studies are necessary in this field before assessing the possibilities of localized subcortical lesions.

SUMMARY

A brief review is presented of personal experiences with 130 patients

operated upon for epilepsy during the last 5 years. In 90 cases the epilepsy was secondary to expansive intracranial lesions. Because of their rarity, tuberculomas, cysticercosis and hydatid cysts are specially mentioned.

In the group of localized cerebral scars the best results were obtained in those cases in which the scars were in the frontal or occipital poles, where wide resections can be performed.

In 12 cases of cerebral hemiatrophy with infantile hemiplegia the epilepsy was treated by partial cortical resection or complete hemidecortication. Six examples of hemispherectomy are reported and in the 4 survivors good results were obtained although 3 still have occasional seizures and contralateral electroencephalographic disturbances.

Anterior temporal lobectomy seems to be a useful treatment for the pure forms of psychomotor epilepsy with an electrical focus in the temporal region (4 cases), but it fails in other epileptics who have generalized convulsive seizures associated with the psychomotor attacks (4 cases).

Stereotaxic lesions of the thalamus were tried in only 2 epileptics. One of these patients, who had a rare form of partial myoclonic seizures, obtained some relief.

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THE DURATION OF NEUROMUSCULAR FUNCTION AFTER NERVE SECTION IN MAN

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CONDUCTION in the distal segment of severed peripheral nerve disappears after a maximum period of 3 to 5 days in a variety of experimental animals: rat, rabbit, guinea pig, dog, cat, and monkey.[3,6,7,9,12–15,19–21,23,26] In contrast, Erb[5] believed that the peripheral segment in man may remain functional for 1 to 2 weeks and Adrian[1,2] and more recent workers[18] have concluded that peripheral neural conduction may survive up to a month after nerve section in man. In better agreement with the results in animal studies are the recent observations by Murphey[16] and Sunderland.[25] The former states that peripheral excitability in sectioned human nerve is lost in 2 to 4 days; the latter, in 90 hours. A subject of such disagreement was considered worthy of special study.

Direct measurement of the nerve action potential is the most nearly accurate method of studying degeneration of conduction when the nerve can be dissected out for recording. This, of course, is impossible in man and percutaneous recording of nerve action potentials would be too insensitive to record small numbers of functional fibers.

Another complication is the fact that neuromuscular conduction fails a day or so before disappearance of the nerve action potential in some species of mammals. Since this dissociation has not been studied in primates, observations upon neuromuscular conduction after nerve section in the monkey were made for comparison with the nerve action potential data of Heinbecker, Bishop, and O'Leary.[9]

Visible and palpable muscle contraction in response to electrical stimulation of nerve has been used in this study as an index of function in the peripheral segment of severed nerve in man.

MATERIAL AND METHODS

Most of the lesions in human subjects studied were the result of lacerations. Direct stimulation of exposed proximal and distal nerve segments was carried out at the time of primary closure in most cases. Subsequently the peripheral segments were stimulated percutaneously at a distance from the muscles innervated. Since most lesions in this study were in the median and ulnar nerves, the intrinsic hand muscles were usually observed with stimulation at the wrist.

A small portable thyratron stimulator was used to deliver 0.1 mfd. condenser charges about once a second. Stimulation at such low frequencies is

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well tolerated (pain fibers respond only at several times maximal intensity for motor fibers[10]), and the identification of direct responses was distinct from the reflex contractions that may be confusing with high frequency stimulation. The indifferent pad electrode was placed conveniently and a 5 mm. disc was the stimulating cathode.

RESULTS

The results in man are summarized in Table 1. Following complete nerve section (Cases 1–5) diminished maximal contraction strength on stimulation of the distal segment was usually apparent 48 hours after the lesion was made. Complete loss of excitability was seen between 91 and 128 hours after section.

TABLE 1

Case History	Nerve	Last Observation of Neuromuscular Response (hours)	First Observation of Complete Inexcitability (hours)
1. Median and medial antibrachial cutaneous nerves cut in upper arm by glass. Major vessels spared.	Median	94	117
2. Median nerve and brachial artery cut at elbow with razor. Extensive blood loss. Tourniquet on arm for 3 hours.	Median	96	107
3. Buccal branch of facial nerve cut in the parotid gland by glass.	Facial	66	91
4A. Median and ulnar nerves cut in middle third of forearm	Median	109	121
4B. by razor. Moderate blood loss from radial artery.	Ulnar	121	128
5. Ulnar nerve and artery cut in lower third of forearm. Moderate blood loss.	Ulnar	72	96
6A. Median nerve cut about 9/10 through by razor. No motor response to stimulation above lesion at time of operation. Moderate blood loss.	Median	95	119
6B. Tourniquet on arm for 3 hours. Ulnar deficit noted postoperatively, presumably caused by tourniquet pressure.	Ulnar	95	119
7. Ulnar nerve cut 4/5 through in lower third of forearm by glass. Muscular response to nerve stimulation above lesion at time of operation. No motor response to stimulation above lesion at elbow the next day.	Ulnar	71	85
8. Median nerve cut almost 1/2 through at elbow with knife. Clinical deficit incomplete.	Median	168	No definite deficiency
Macaque. Nerves aseptically severed in arm.	Ulnar	72	89
	Median	54	72

Two nerves (Cases 6A and 7) were not divided entirely across but showed the same loss of electrical excitability seen with anatomically complete sections. This indication that all fibers had degenerated in spite of partial gross continuity of the nerve was confirmed by the subsequent rate of recovery. No significant excitability changes occurred in a less severe partial transection (Case 8).

In one patient (Case 6B) ulnar paralysis apparently resulted from tourniquet pressure on the arm during surgery. The patient was in a schizophrenic panic state and reliable examination was difficult. During exploration and primary suture of the median nerve in this patient (Case 6A) the intact ulnar nerve was identified beneath a partially severed flexor carpi ulnaris muscle. Postoperatively an ulnar deficit was noted. The ulnar became inexcitable at both elbow and wrist after 119 hours. It was impossible to follow this patient closely, but it is certain that the ulnar did not recover in the next 2 or 3 weeks. Nine months later ulnar function was clinically perfect; there was a moderate median deficit. It is inferred that the ulnar nerve underwent complete Wallerian degeneration and subsequently regenerated.[22,24]

The ulnar and median nerves were aseptically severed in the upper arm in one adult macaque monkey (Table 1). The median was unresponsive at 72 hours and the ulnar, at 89 hours. This correlates fairly well with the disappearance of the alpha spike in the nerve action potential of the macaque[9] and indicates that failure of endplate conduction occurs at approximately the same time as loss of nerve conduction. It would appear likely that this relationship is similar in man.

DISCUSSION

The results indicate that neuromuscular function in man as in other mammals disappears 3 to 5 days after nerve section. Denervation is certainly complete by the end of the 6th day. Therefore the distinction between complete Wallerian degeneration and less severe injury can be made on the basis of the disappearance of excitability in the peripheral nerve segment at this time.

It is probable that when stimulation has to be applied close to the site of traumatic or surgical manipulation, premature loss of neural conduction may occur. In these cases such local effects of trauma are considered insignificant since there is no positive correlation between functional survival time and the distance from the lesion to the point of stimulation. Three lesions were more than 30 cm. from the area tested.

The disagreement with earlier workers may be attributed to a difference in the clinical material they studied. Apparently Erb's[4,5] data are largely based upon observations in nontraumatic neuropathies and late traumatic lesions. Similarly Adrian's[1,2] cases did not include acute traumatic nerve sections. His often reproduced diagram of the progressive change in motor point strength-duration curve after nerve section is derived from a case of Bell's palsy, in which degeneration can scarcely be assumed to be as acute or as complete as in nerve severance. Moreover, his conclusions are based not upon nerve stimulation, but upon a single discontinuity in the motor point strength-duration curve, and recent observers[17] have not found the single discontinuity uniformly.

It follows from the present study that all changes in motor excitability

from the end of the 6th day onward must be the result of progressive excitability changes in the muscle fibers alone. Since the question of clinical significance is whether or not nerve function exists, it seems unnecessary in most cases to study secondary changes in muscle excitability in order to establish the diagnosis of denervation.

The technique described here requires any simple source of current and no special training. With the addition of a shielded needle electrode, study can be extended to nerves that are not readily accessible to superficial stimulation and to nerves that are injured near their terminations. The loss of muscle twitch where contraction was previously present is a sharp endpoint. By contrast it may be noted that the most elaborate muscle stimulation apparatus still depends upon the determination of threshold contraction by the fallible eye and hand of the observer.[14] Moreover, changes of excitability as time goes by are gradual rather than distinct.

Electromyography and muscle excitability changes are reported to be of predictive value in recovering nerve injuries, but their complexity prevents their wide application. Direct stimulation of the proximal portion of regenerating nerves has been combined with electromyographic recording by Harvey and Kuffler[8] and by Hodes, Larrabee, and German.[11] It may be that such synchronous activation of nerve would give perceptible movement of predictive value before gross clinical signs of recovery appear.

SUMMARY

Electrical stimulation has been applied to the peripheral portions of human nerves after traumatic section, and to macaque nerves after surgical section. As in other mammals, motor function is completely abolished 3 to 5 days after the lesion is produced. The muscle is therefore certainly completely denervated by the end of the 6th day.

The technique used in this study is proposed as a simple direct method of establishing the diagnosis of complete Wallerian degeneration after nerve injury.

I am indebted to the Surgical House Staff of St. Louis City Hospital for their generous cooperation.

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CASE REPORTS

CEREBRAL CALCIFICATION EPILEPSY

REPORT OF A CASE OF EPILEPSY CAUSED BY A CALCIFIED HAMARTOMA OF THE BRAIN

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The radiographic demonstration of calcified shadows in the cerebral hemispheres of persons undergoing investigation for epilepsy always attracts attention. One such case is reported in this communication and is of interest in that it resembles very closely the rare condition described by Geyelin and Penfield[1] as "cerebral calcification epilepsy."

CASE REPORT

A male aged 40 years, referred by Dr. John Willis, was admitted to the Neurosurgical Unit, Dunedin Hospital, on June 30, 1949, with the chief complaint of epileptic seizures for 35 years. Between the ages of 5 years and 14 years epileptic seizures had recurred daily. During these he had lost consciousness and occasionally passed urine. The seizures lasted about 15 minutes but his recollection of the details was vague. After the age of 14 years the attacks had diminished in frequency, and by the age of 16 had disappeared.

At the age of 19 years, the fits recommenced. Each attack began with a drawing sensation in the left side of the face and a feeling of numbness, spreading from the left side of the forehead to the right. This was followed in a few seconds by loss of consciousness. The attacks now were of shorter duration than those occurring in the earlier period. The frequency had gradually diminished from once in a week to once in 2 or 3 months. Although he would fall if standing when a fit began, no convulsive movements occurred.

Family History. The patient was the seventh and youngest child in his family. The child preceding him was stillborn, while the fifth child had died at the age of 10 weeks. Three of his elder brothers had died in adult life from causes given as: "double pneumonia," "1918 influenza epidemic" and "death on active service." None of his siblings, neither of his parents, and none of his uncles and aunts had any history of seizures. There were, however, two known instances of epilepsy in male relatives. A maternal cousin with epilepsy had been admitted to a mental hospital at the age of 19 years and died in status epilepticus at the age of 38. In his case there is no record of any X-ray of the skull nor was there a postmortem examination. His history suggests that he always fell on his left side and that his fits were preceded by a headache over the eyes. He was mentally retarded. A second male relation, a grandson of his mother's youngest brother, suffers from epilepsy but no details are available. It is therefore possible that a hereditary factor may have operated in producing this patient's symptoms.

Examination. His mentality was rather childish. He lived with an aged mother and, although he did the usual household chores and attended to the garden, he had never held a steady job. When crossed he became argumentative, but he relied on others to lead him. He was a well-nourished man, alert and cheerful but of only moderate intelligence. He was right-handed. His speech was jerky but there was no aphasia. A scar was present on the right side of the forehead, a result of falling upon a heater during a fit sometime before. There was slight nystagmus on extreme deviation of eyeballs to either side. The visual fields were full.

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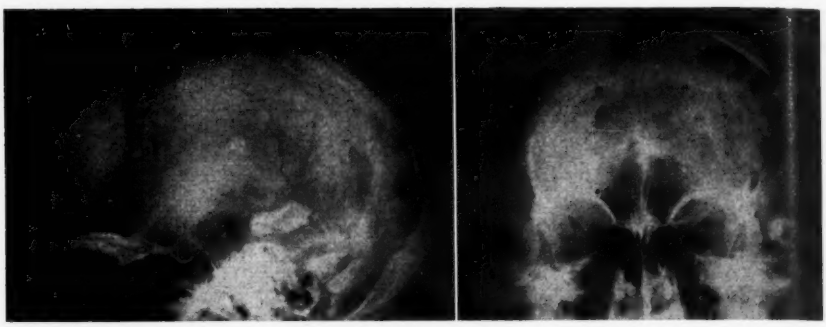
Blood Wassermann and Kahn tests were negative. CSF pressure was normal; the fluid contained no cells and 40 mg. of protein/100 cc. X-rays showed an elongated calcified shadow, 3.1 cm. × 1.3 cm., in the right temporal lobe (Fig. 1) which in the axial view (Fig. 2) was in the shape of a crescent, with concavity outwards. There was also a small and indistinct shadow, 0.8 cm. in diameter, in the left parietal region (Fig. 1). Air encephalography disclosed a normal ventricular system. The right temporal horn lay immediately deep to the calcified shadow and was not distorted by it.



FIGS. 1 and 2. (*Left*) Lateral radiograph of skull showing ovoid calcified shadow in right temporal lobe and smaller rounded shadow in left parietal region. (*Right*) Anteroposterior view showing the crescent-shaped calcified area in right temporal lobe near the surface. The second smaller calcified shadow is not visible in this film.

Course. Shortly after admission, a major epileptic fit was observed in the ward. This began with a hoarse cry. The patient was seated with eyes open and fixed, and breath held. The right arm then began to twitch and he slid forward in the chair. Within a few seconds, the twitching had spread to his left side. The movements were not violent nor extensive. He remained unconscious for 4 minutes. This was the only occasion on which twitching was observed on the right side.

Although a definite relation between the calcified shadow and his fits was not established, it was presumed that the sensory aura in the left side of the face was associated with the right temporal lesion. The patient was obsessed with the fear of further epileptic attacks and he demanded operation.

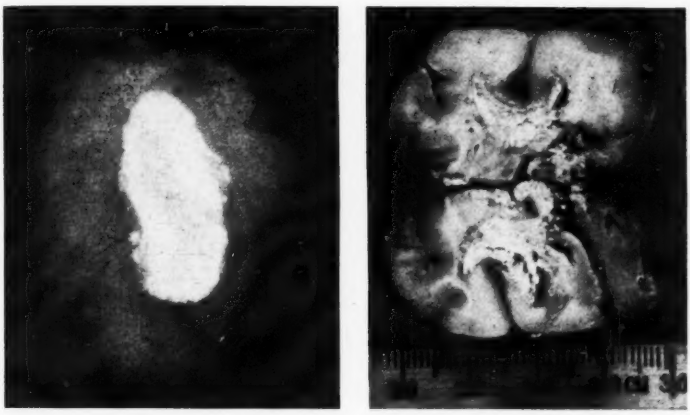
Operation (M.A. Falconer). A right lateral craniotomy was performed under local anaesthesia. The surface of the exposed brain appeared normal, although the gyri were perhaps small and the sulci wide. In the inferior margin of the temporal lobe a bony-hard lesion could be felt immediately beneath the surface, although the overlying cortex itself did not appear to be abnormal. The area containing this lesion was removed in a block about 4 cm. in antero-posterior extent and 2.5 cm. deep.

His *postoperative course* was straightforward. Examination, however, disclosed a left, upper quadrantic, homonymous hemianopia.

Subsequent Course. When seen 8 months after operation he was apparently well and free from seizures. In March, 1952, 2½ years after operation, he stated that apart from a single seizure 9 months after operation, he had remained free from symptoms for 1½ years. In the following year he had had two episodes in which several seizures occurred within a few days. The patient himself claimed that he felt fitter since the operation and that his seizures were much less frequent. Examination at this time revealed no significant features. X-rays of the skull in June, 1951 demonstrated the area of calcification in the left parietal region to be unchanged.

Pathological Report. The tissue removed at operation consisted of a cube of cerebral cortex

and white matter. X-ray of the block showed the zone of calcification clearly (Fig. 3). The greater density of the margin was produced by the crescentic-shaped distribution of the calcium. On section (Fig. 4) the sulcus which ran across the centre of the segment was seen to gape in its deepest part. In the white matter around this gaping sulcus was a zone of dense calcification extending up on either side of the sulcus about half way to the surface. The depth of the calcified zone was no more than 5 mm. The cortex at the bottom of the sulcus was narrowed.



FIGS. 3 and 4. (*Left*) X-ray photograph of specimen after removal. (*Right*) Cut surface of block of cortex and white matter removed at operation showing the gaping sulcus and crescentic mass of calcific material in the white matter.

Microscopic Examination. There was an extensive deposition of calcium in the white matter immediately beneath the cortex. This was arranged in collections varying in size from small granules (Fig. 5) to large solid lamellae (Figs. 5 and 6). Many of these lamellae had a concentric laminated appearance which suggested that they had developed around blood vessels which had since become occluded. Calcified plaques were present in the walls of some of the vessels still patent, and involved both media and intima. The white matter surrounding the calcified area showed intense gliosis which extended through the whole thickness of the cortex in the bottom of the sulcus. The cortex lining the walls of the sulcus was normal; the transition between this normal cortex and the scarred cortex in the floor of the sulcus was abrupt.

Comment. The lesion appears to be primarily a calcification in the walls of blood vessels with scattered calcium granules in the surrounding tissue. Although the junctional zone between cortex and white matter is normally very vascular, the number of blood vessels apparently present in this lesion is much greater than normal; this suggests that an abnormal collection of blood vessels must have preceded the deposition of calcium.

DISCUSSION

This case resembles very closely the condition described by Geyelin and Penfield[1] and called by them "endarteritis calcificans cerebri." In their series, the patients belonged to one family and included the father and all four of his children. Each of the patients had multiple discrete calcified foci within the brain, many of which gave the impression of being hollow and all of which were apparently superficial. In the one child (Case 3) who was operated on, a crescentic zone of calcification was

found in the depths of a sulcus, the gross specimen appearing very similar to that described in this report. The microscopic features were also similar, calcification beginning in the deeper layers of the gray matter and extending into the white matter. The calcium was found in the form of solid casts or discrete granules about the larger vessels. The deposits most frequently occurred in the media. The lumen of the vessels was often found decreased to the vanishing point. There was extensive gliosis in the surrounding tissue. This child improved for a time after operation, then, because of recurrences of seizures, periarterial sympathectomy of the carotid and vertebral arteries was carried out with benefit.

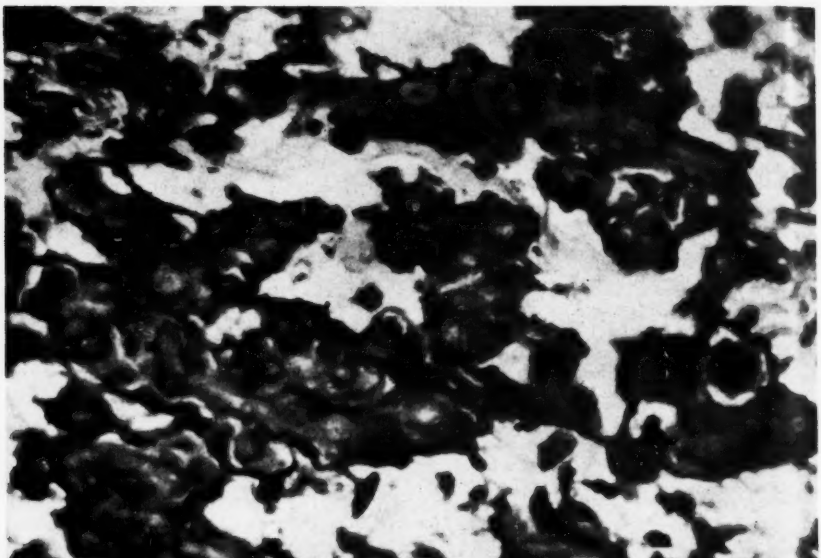


FIG. 5. Photomicrograph showing concentrically calcified lamellae. Hematoxylin and eosin, ×260.

Geyelin and Penfield suggested that the primary pathological process was probably a slowly progressive closure of the end arteries in the cerebrum which produced an area of local necrosis chiefly at the level at which the short pial vessel supply ends and the deeper blood supply begins. This area of degeneration then became secondarily calcified, and calcium was laid down in the walls of the involved small vessels.

In a series of cases of calcifying epileptogenic lesions reported by Penfield and Ward[2] 5 examples of hemangioma calcificans were found, each occurring within one temporal lobe and situated close to the fissure of Sylvius. The two photographs of the gross lesions and the three photomicrographs in their paper do not resemble those of the present case.

The lesion described here differs from the racemose angioma and from the an-

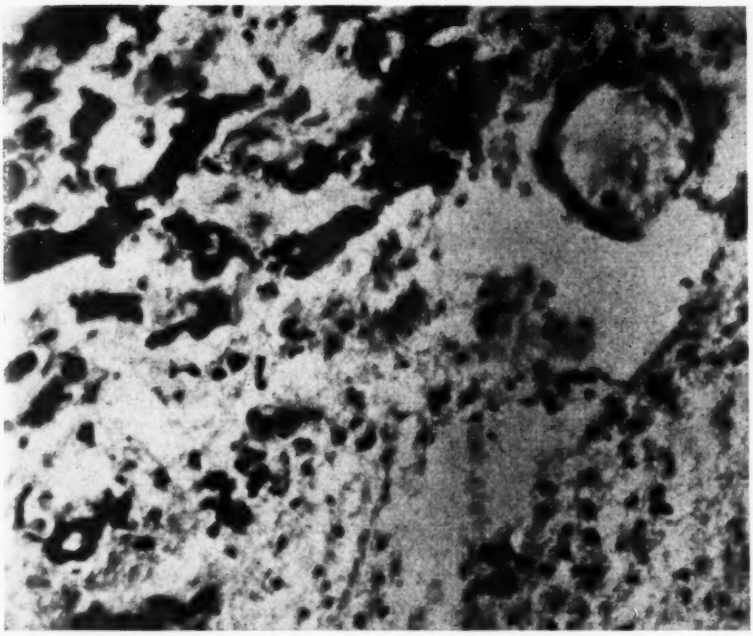


FIG. 6. Photomicrograph showing deposition of calcium around capillary blood vessels and in the wall of a larger vessel cut in cross section. Some calcific granules are present in the surrounding white matter. Hematoxylin and eosin, ×260.

giomas of the Sturge-Weber type. The differences from these two more common lesions and from Penfield and Ward's "hemangioma calcificans" justify a separate classification, especially since the lesion is multiple and apparently familial.

In the case described here it was felt that the condition had developed as a degenerative change in an existing vascular abnormality and the alternative name suggested is "calcified subcortical hamartoma."

SUMMARY

1. A case is reported in which a calcified lesion in the temporal lobe of a chronic epileptic was discovered on X-ray examination and removed surgically.

2. Pathological examination showed the lesion to consist of a mass of blood vessels, nearly all of which had depositions of calcium in their walls, leading in some instances to the complete obliteration of the lumen of the vessel. The lesion is therefore regarded as a calcified hamartoma.

3. Similar conditions reported in the literature are discussed, with emphasis on the resemblance between this lesion and that described by Geyelin and Penfield as "endarteritis calcificans cerebri."

I have to thank Mr. Murray Falconer, F.R.C.S., under whose care the patient was treated in hospital, for access to clinical records and assistance in the preparation of this paper; and Mr. E. R. Macdonald, who carried out the photographic work.

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A CASE OF BRAIN TUMOR WITH UNILATERAL ELEVATION OF RETINAL ARTERY PRESSURE

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The determination of retinal artery pressure may be a helpful diagnostic procedure in cases presenting cerebral vascular problems. A significantly lowered retinal artery pressure is present on the side of the involved internal carotid artery in cases of thrombosis of the internal carotid artery, and the procedure is a reliable and valuable aid in the differential diagnosis of this condition. In a group of 7 patients with proven thrombosis of the internal carotid artery there was an average per cent difference of 31.7 per cent diastolic and 24.1 per cent systolic pressure as compared to 5.2 per cent and 3.3 per cent in normal controls.[3]

Recently we have had the opportunity of observing a case that illustrates the possibility of misinterpreting retinal artery pressure determinations. The ophthalmodynamometric findings in this case, while not entirely typical of thrombosis of the internal carotid artery, certainly might lead to such a diagnosis. The case is, therefore, reported briefly in order that those working with the technique of ophthalmodynamometry may become cognizant of the occurrence of this unusual retinal artery pressure variant and thus avoid misinterpretation of the readings.

CASE REPORT

A 49-year-old white male had sudden onset of difficulty in verbal expression in June, 1951. At first this difficulty lasted about 2 hours, but it had recurred episodically since that time, and for the 2 weeks prior to his admission it had become almost constant. He also had noted difficulty in writing and typewriting, and occasional mental confusion. For the last 4 days there had been an abnormal sensation on the right side of his face. He had had no weakness of either upper or lower extremities and no urinary difficulty. Headache was not a complaint.

Examination. The patient was right-handed. B.P. was 140/80. The fundi and visual fields were normal. There was a right supranuclear facial palsy. The uvula deviated slightly to the left. There was some deviation of the tongue to the right. Deep tendon reflexes were slightly overactive, but symmetrical except for a slight increase of the right ankle jerk. There was a marked expressive aphasia. Blood and urine were normal.

X-rays of the skull and chest were normal. An EEG indicated a lesion in the left posterior frontal and lower motor and midtemporal regions. No true neoplastic waves were seen. The EEG was believed to support a diagnosis of degenerative disorder rather than neoplasm. CSF pressure was 150 mm. of water; the fluid was clear and contained 32 mg./100 cc. of total protein; Kahn test was negative; globulin, colloidal gold and mastic tests were normal.

Retinal Artery Pressure Determinations. On Sept. 12, 1951 there was a systolic pressure of 58 gm. of water with a diastolic of 10 in the left eye, and over 150 diastolic pressure in the right eye. The systolic pressure on the right could not be measured. On Sept. 14, 1951 the retinal

artery pressures were unchanged. After a stellate block on the left side there was no change in the retinal artery pressure reading on the right, but the pressure in the left eye had risen to 90 systolic over 32 diastolic. This response to blocking of the cervical sympathetic chain was thought to substantiate the diagnosis of thrombosis of the left internal carotid artery.[2]

Course. A left percutaneous arteriogram was done which showed no evidence of vascular occlusion. As the patient's condition was becoming progressively worse, ventriculography was performed Sept. 24, 1951. This showed compression of the left lateral ventricle, and shift of both lateral ventricles and the 3rd ventricle to the right, without shift of the posterior ventricular structures, suggesting a frontoparietal space-occupying lesion on the left. A craniotomy was performed, and a large surface neoplasm was encountered which was partially removed. Despite several ventricular taps to reduce pressure, the patient died on Sept. 25, 1951.

Pathological Study. The tumor was a glioblastoma multiforme involving the left frontal and parietal lobes. There was marked generalized cerebral edema.

DISCUSSION

It is to be noted that a systolic pressure determination could not be made in the right eye. This is in itself unusual and not characteristic of the findings of thrombosis of the internal carotid artery. Unless the ophthalmodynamometric findings are completely typical of those reported in a series of verified internal carotid artery thromboses[3] they should be viewed with some suspicion and further diagnostic steps should be taken. In this case arteriography conclusively ruled out the possibility of a left internal carotid artery thrombosis, and it became necessary to perform a ventriculogram in order to finally establish the diagnosis of a space-occupying lesion.

It is interesting to speculate as to the possible relation of such marked retinal artery pressure differences to the future development of unilateral papilledema or even to the Foster Kennedy-Gower syndrome.

The present case raises the question of the relationship of retinal artery pressure to papilledema. Bailliart[1] showed that in papilledema retinal artery pressure was elevated. In the case under discussion it seemed logical to surmise that optic atrophy might have developed in the eye with the significantly lowered retinal artery pressure and thus led to the development of the Foster Kennedy-Gower syndrome.

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MEDULLOBLASTOMA WITH ONSET DURING THE NEONATAL PERIOD

REPORT OF A CASE

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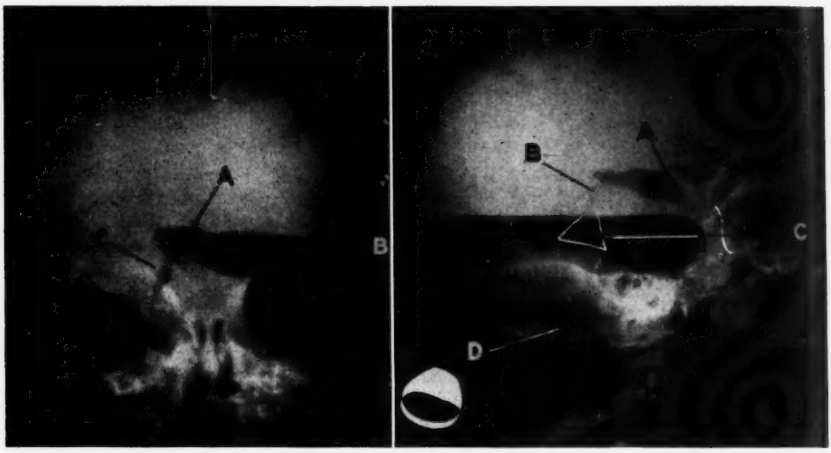
(Received for publication July 14, 1952)

Medulloblastomas that produce symptoms during the first two months of life are exceedingly rare. Arnstein *et al.*,[1] when reporting an instance of an intracranial tumor in a 3-day-old infant, could find only 13 recorded cases in which symptoms of

a tumor appeared before the 60th day of life. More recently, Thiele and Dimmick[6] described an infant who at birth had hydrocephalus which was later found to be the result of a glioma. These authors reported 3 other instances of infants with primary intracranial neoplasms which produced symptoms in the first few weeks after birth. Iyer[4] reported an adamantinoma in a newborn, which is almost unique. Of the 19 tumors recorded in the three papers, only 2 were thought to be medulloblastomas, 1 occurring in the right cerebral hemisphere and 1 about the iter.

The infant whose case history is recorded below first manifested symptoms of an intracranial neoplasm during the second week of life. Undoubtedly the tumor arose before birth.



FIGS. 1 and 2. Ventriculograms taken with child suspended by heels. *Left*, (A) Dilated 3rd ventricle. (B) Air in temporal horn demonstrating marked hydrocephalus. (C) Aqueduct of Sylvius deviated to right side by a mass in the cerebellum. *Right*, Lateral view. (A) 3rd ventricle. (B) Aqueduct of Sylvius. (C) Deformed 4th ventricle, retouched. (D) Air in cisterna magna, indicating incomplete obstruction of 4th ventricle.

CASE REPORT

A 3-month-old infant was admitted to the hospital with the diagnosis of hydrocephalus. It had been born at this hospital on July 30, 1950, after a normal prenatal course and labor. Examination at birth and during its stay in the hospital had disclosed no abnormalities. The birth weight was 9 lbs. and 12 oz.; the head measured 15 in. in its greatest circumference; the chest measured 14½ in.; and x-ray of the chest was normal.

During its second week of life, projectile vomiting began, so violent that vomitus was frequently projected out of the bassinet. Pyloric stenosis was suspected, feedings were changed and atropine was given. Despite these measures the child continued to have paroxysms of coughing followed by violent vomiting. Otherwise it remained well but gained weight slowly.

At 3 weeks of age the baby could roll over by itself. When it was 8 weeks old, however, it no longer attempted to raise its head or roll over, but lay quiet and placid in its crib. The mother first noticed the child's head was rapidly enlarging when it was 10 weeks of age. The physician who had been seeing the infant frequently was insistent that there had been no evidence of hydrocephalus until this time, and that both fontanelles had been small and depressed.

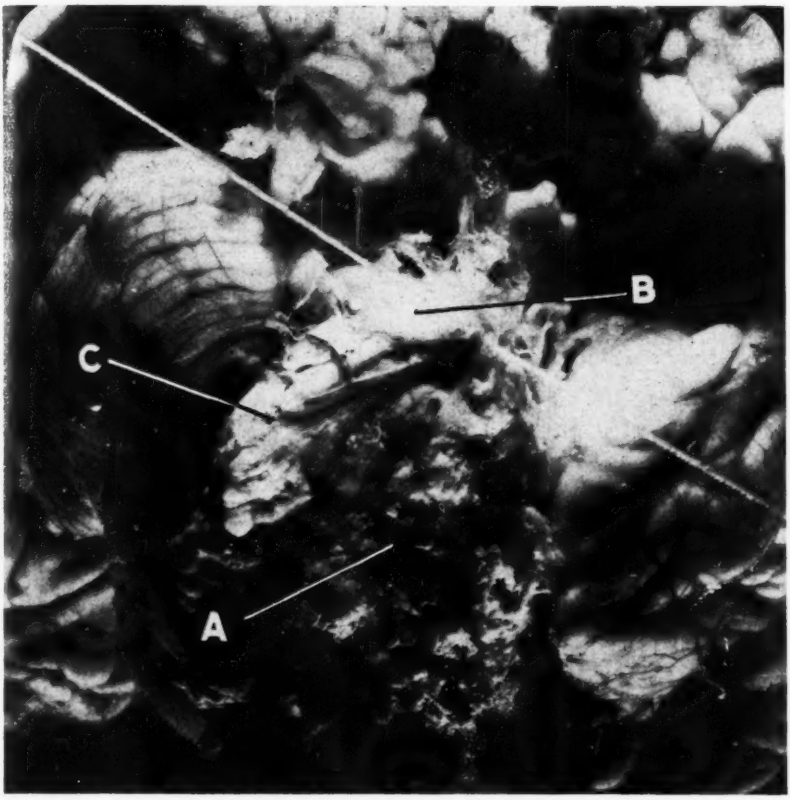


FIG 3 View of base of brain. (A) Necrotic left cerebellar hemisphere; note filling of region of left cerebellopontine angle by tumor. (B) Flattened pons. (C) Deviated and distorted medulla.

Examination. The child was well developed and well nourished despite the long history of vomiting. It appeared in no acute distress. Its weight was 13 lbs. The head measured 18 in. in circumference and had a globular appearance. The anterior and posterior fontanelles were open widely and were bulging. The scalp veins were moderately dilated. Temperature was normal; urine and blood were not remarkable; Wassermann reaction was negative.

Ventriculography was done through the anterior fontanelle, using a small amount of air. The lateral and 3rd ventricles were symmetrically dilated. The aqueduct of Sylvius was also dilated but was displaced to the right. The 4th ventricle was deformed by an extrinsic mass in the left cerebellar hemisphere. The ventricle was not blocked, however, and air passed into the cervical canal (Figs. 1 and 2).

At the same time phenosulfonphthalein was placed in the lateral ventricle and was recovered from the lumbar canal 20 minutes later, indicating a communicating type of hydrocephalus. Analysis of the ventricular fluid showed 5 lymphocytes/c. mm. and 65 mg. per cent of protein. The next day an intravenous pyelogram was done; the kidneys appeared normal. The child had a fever with temperature up to 100.3° which fell to normal 8 days later. It was thought by the physicians who saw the child that there was no evidence for pyloric obstruction.

Operation. To control the hydrocephalus, a spinal-ureteral anastomosis, as described by Matson,[5] was done.

Course. Four days after operation the head measured $17\frac{3}{4}$ in. The fontanelles were shrunken and the cranial bones were now overlapping. For a few days the child seemed to eat better but still continued to vomit at irregular intervals. Then it became listless, vomited more frequently and it was with difficulty that the electrolytic balance was maintained.

Eight days after operation, the left ear showed signs of an otitis media. Penicillin had been administered since operation, but despite this there had been a slight elevation of temperature and the WBC had varied between 10,000 and 20,000 cells/c. mm. A myringotomy was done and a few drops of pus were obtained. Two days later a complete left facial paralysis of peripheral type was noticed. There was also paralysis of the left side of the pharynx. It was assumed that the mass in the cerebellar hemisphere had extended to involve the cranial nerves at the cerebellopontine angle.

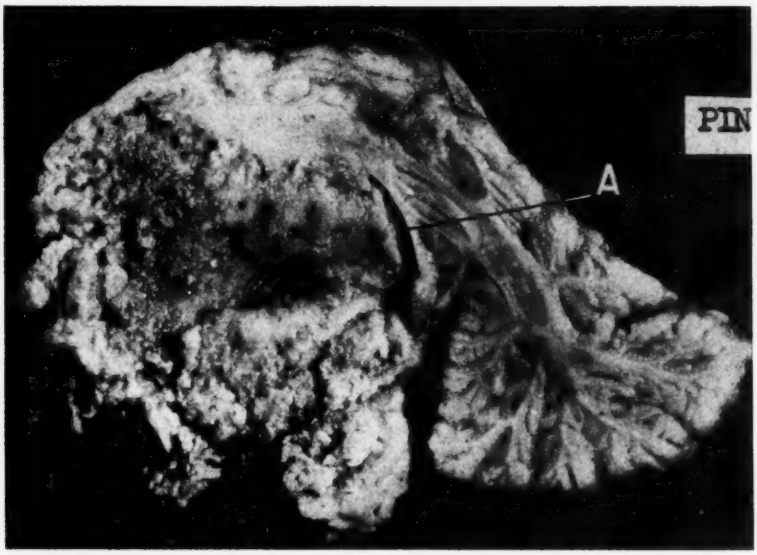


FIG. 4. Section through cerebellum demonstrating extensive destruction of left cerebellar hemisphere by the neoplasm. (A) Displaced and deformed 4th ventricle.

From this point on, the child began to deteriorate. The left corneal reflex was lost, followed by disappearance of the cough reflex. The infant no longer cried and large amounts of secretions accumulated in the nasopharynx. Since it was assumed that the intracranial mass was caused by an extension of the infection in the middle ear, and since penicillin had been of no benefit, chloromycetin and aureomycin were tried.

About 3 weeks after operation, the fontanelles became tense and the head re-expanded to $18\frac{3}{4}$ in. The spinal-ureteral anastomosis was revised and the hydrocephalus was once more controlled. There was no change in the baby's general condition, and it expired on Dec. 11, 1950 at the age of $4\frac{1}{2}$ months.

Autopsy. There was an aspiration pneumonia. In the left cerebellar hemisphere was a large necrotic mass which looked like an infected infarct (Fig. 3). An obliterative arachnoiditis had occurred about the medulla and lateral cisterns, which accounted for the hydrocephalus. When the brain was sectioned, the entire ventricular system was seen to be enormously

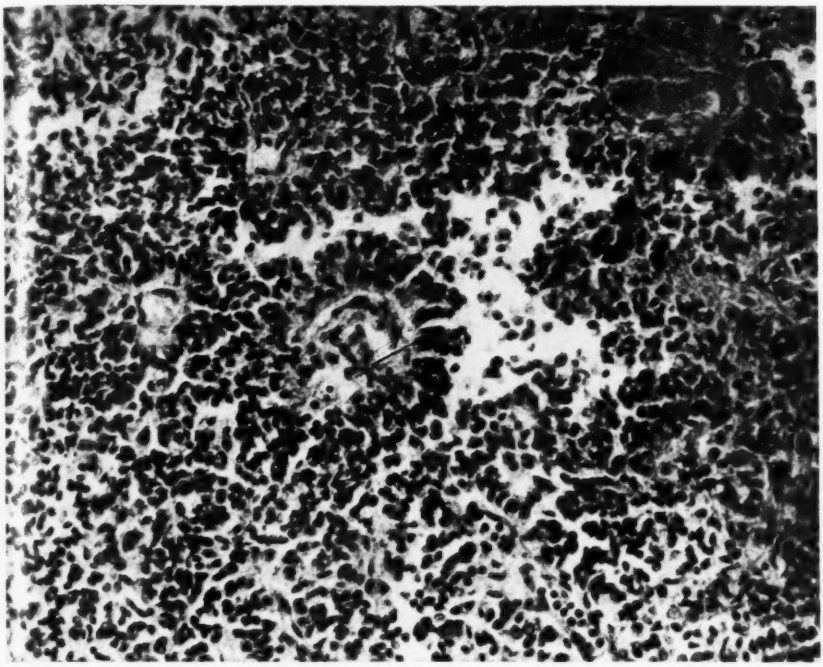


FIG. 5. Low-power magnification of tumor. Note pseudorosette formation, especially about blood vessels.

dilated. The cerebral cortex was only a few millimeters in thickness, while the floor of the 3rd ventricle was no thicker than tissue paper. The ventricular system was patent. The left lobe of the cerebellum was filled by a soft necrotic mass which depressed the 4th ventricle, and which had extended laterally to fill the cerebellopontine angle (Fig. 4). There was no evidence of an infection in the left middle ear, or involvement of the petrous ridge by osteomyelitis. The dural sinuses were normal.

Microscopic Examination. The necrotic mass in the left cerebellum was a rapidly growing neoplasm. It was made up of small cells with scant cytoplasm. The size of the nuclei varied considerably and many mitotic figures were present. The nuclei stained darkly and were granular. Much of the tumor tissue was necrotic and amorphous. The cytological pattern was characteristic of a medulloblastoma. Invasion of the medulla and subarachnoid space about the cerebellopontine angle had occurred (Figs. 5 and 6).

COMMENT

From the available literature, it is clear that reports of intracranial tumors appearing at birth or during the neonatal period are rare, and that the antemortem diagnosis has seldom been made. In only 3 of the recorded cases was the diagnosis suspected before death (Holt,[3] Cushing,[2] Thiele and Dimmick[6]). It should also be mentioned that in contradistinction to the situation in older children, of the 19 reported cases of brain tumors occurring in this age group, only 5 were infratentorial and over one-half were gliomas.

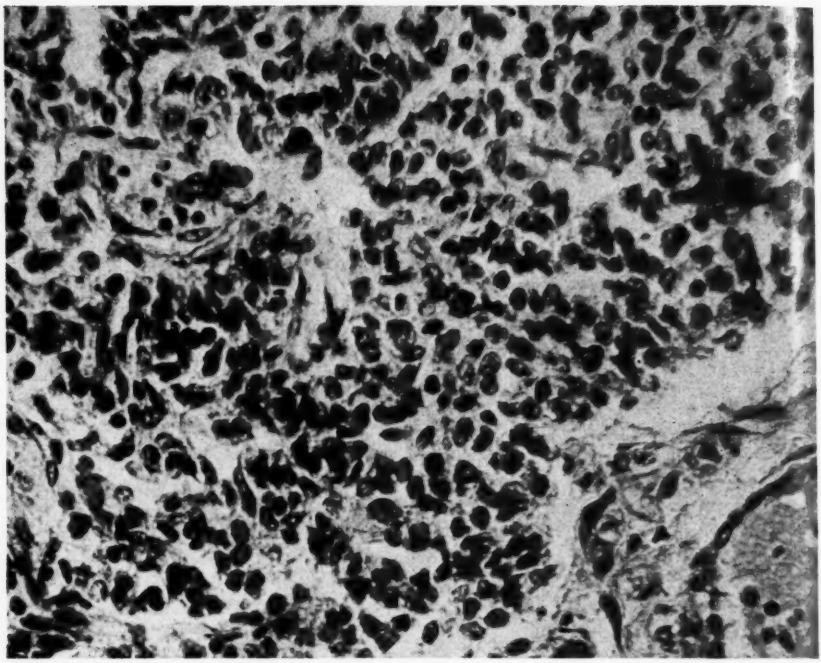


FIG. 6. High dry magnification. The cells are clumped about blood vessels. There is variation in size of cell nuclei, and many mitotic figures are present. Scanty cytoplasm of cells is evident.

The symptomatology of brain tumors occurring in this period is often obscure and ill defined. Hydrocephalus is the most frequent finding. In the case reported it was during the study of what was thought to be hydrocephalus on a developmental basis that a mass in the cerebellum was found. Vomiting was the only symptom that was unusual for hydrocephalus. It was much later that additional findings pointed to a mass in the left cerebellopontine angle. The routine use of air studies of hydrocephalic infants would no doubt disclose more instances of brain tumors during this age.

Because of the failure to evaluate the symptoms and findings fully, the treatment in this case left much to be desired. The vomiting was a significant symptom, since there was no evidence of pyloric stenosis and vomiting is most unusual with hydrocephalus. The presence of 65 mg. per cent protein in the ventricular fluid should have aroused more suspicion as the normal value even with hydrocephalus is rarely more than 15 mg. per cent. Finally, the demonstration of a mass in the left cerebellum should have led to an early cerebellar exploration.

Since air and dye rapidly entered the cisterna magna but did not circulate along the base of the brain and over the hemispheres, it was thought that the hydrocephalus should be controlled first. This did not significantly improve the infant's condition. When the otitis media was discovered in the left ear, it was immediately assumed that the mass in the cerebellum was an abscess, and that the hydrocephalus

was caused by a basilar arachnoiditis. While I had never encountered this situation in a child so young, it seemed a logical assumption at the time. That the infected ear was an adventitious finding, and that cerebellar neoplasm might be obliterating the basilar cisterns was not thoroughly considered simply because of the child's age.

CONCLUSIONS

While brain tumors during the neonatal period of life have rarely been reported, they should be kept in mind when evaluating infants with signs and symptoms of an intracranial disorder. Persistent vomiting should direct suspicion to the head when a clear-cut demonstration of gastro-intestinal pathology is not present.

In hydrocephalic infants who vomit persistently and who have an increased protein content of the ventricular fluid, a tumor is very likely, and an air study should be carried out. Using a small amount of air, the procedure is safe. A greater awareness will undoubtedly lead to more frequent antemortem diagnosis.

SUMMARY

There has been presented a third reported case of a medulloblastoma occurring in an infant of the neonatal period. It appears to be the first reported instance in which a posterior fossa neoplasm was demonstrated by means of a ventriculogram in a child of this age. Findings that suggest intracranial tumors in early infancy are discussed.

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INTRADURAL LIPOMA OF THE SPINAL CORD

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Two reports have appeared in the recent literature describing cases of intradural spinal lipoma.[2,3] In both cases operation was performed, but the tumor appeared to be intramedullary and so only a palliative procedure was done. About the time these articles appeared, a similar case was observed at the University Hospital. When surgically exposed, the tumor was thought to be intramedullary, but further study

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suggested its benign, extramedullary nature so that at a second operation, a successful attempt at total resection was made. Because the second operative procedure revealed the true extramedullary position of this lipoma, it was agreed that a report of the case with detailed operative findings was indicated.

A review of the literature concerning intradural spinal lipoma by Ehni and Love[1] in 1945 listed 29 detailed cases and mentioned 10 or 12 more in which insufficient data were given. Of all these cases, in only 5 was the tumor surgically resected: 1 by Root, 1 by Sachs and Fincher, 2 by Elsberg, and 1 by Ehni and Love. Eight more cases were reviewed by Johnson,[2] in 4 of which resection was performed. Taniguchi and Mufson[3] reported 1 case in which the tumor was not resected, bringing the total to over 48. The case presented here, we believe, is the ninth reported case in which an intradural lipoma of the spinal cord was totally resected.

CASE REPORT

M.J., a 31-year-old negro female, was first examined in the out-patient department of the University Hospital in August, 1950.

She dated the onset of her present illness to April, 1950. At that time, while 8 months pregnant, she had experienced back pain in the low thoracic and lumbar regions and noted weakness in the left leg and numbness of the left foot. Following delivery, there was a steady progression of weakness, stiffness, and numbness which began to involve both lower extremities. During the last month of her illness, she could not walk without support. She also had nocturia of 2–3 times and urgency of urination. In the first week of August, 1950, she had burned her buttocks with a hot water bottle without any sensation of pain.

Examination. The patient was an apparently healthy colored female. Tenderness was noted on pressure over the spinous processes of the lower thoracic vertebrae. The left leg was 1–2 cm. smaller in circumference than the right at all points measured. There was about 60 per cent loss of motor power in all muscle groups of the left leg and about 30 per cent loss on the right side. Moderate increased resistance to passive motion was noted, more on the left than the right. Superficial abdominal reflexes were absent. Patellar and Achilles tendon reflexes were markedly hyperactive in both legs. The response to plantar stimulation was flexor on both sides. There was diminished pain and thermal sensibility throughout all the lumbar and sacral dermatomes on both sides and hyperesthesia in the 12th thoracic dermatomes bilaterally. Position sense was lost in both lower extremities. Walking was extremely difficult even with a cane and was accomplished on a wide base by keeping the knee and ankle joints stiff.

Roentgenograms of the chest and spine were interpreted as showing no abnormality. Blood serological test for syphilis was negative. Clinical impression: lower thoracic spinal cord tumor. Myelography confirmed the presence of an intraspinal tumor.

1st Operation, Oct. 17, 1950. The spinous processes and laminae of the 7th, 8th, 9th, 10th and 11th thoracic vertebra were removed. The dura was incised and a large yellow mass protruded into the opening. This mass was apparently in the spinal cord with the pial vessels continuous over it. The swollen cord filled the entire canal so that the dorsal roots could not be visualized. Normal spinal cord was seen above and below the mass, which measured about 10 cm. in length. At the site of the tumor itself, however, no demarcation from recognizable spinal cord could be seen. A dorsal midline incision was therefore made into the yellow mass. Several pieces of tumor were removed by sharp dissection, without any evidence of neural tissue. The tumor was firm and homogeneous but appeared spongy on the cut surface. The dural defect was covered with a saline-moistened Gelfoam® and the wound was closed without drainage.

Postoperative Course. There was no motion in the lower extremities for the first 5 days and there was complete loss of bladder control for the first week. She improved rapidly and had regained her pre-operative neurological status at the end of 4 weeks.

Pathologic Examination. Gross. The fragments of tumor removed were soft, slightly fibrous and yellowish. The cut surface was homogeneous, glistening, and fatty.

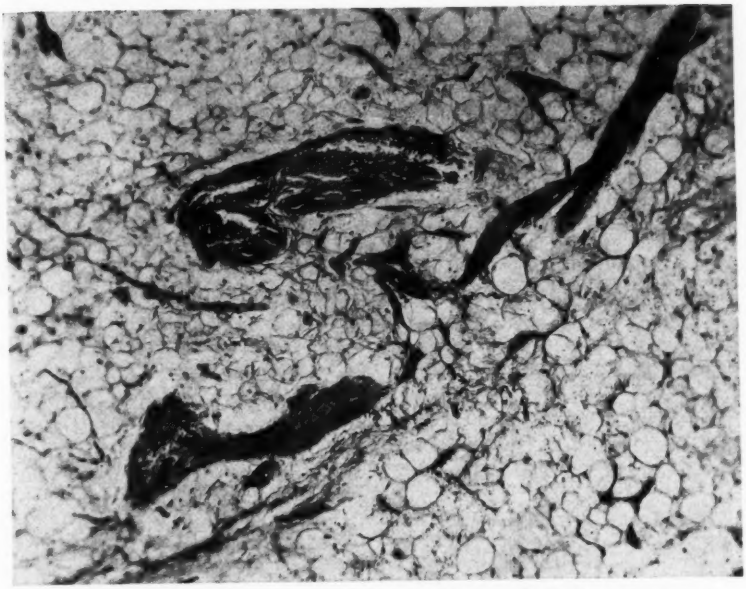

FIG. 1. Photomicrograph of degenerative nerve roots in the midst of the adipose tissue

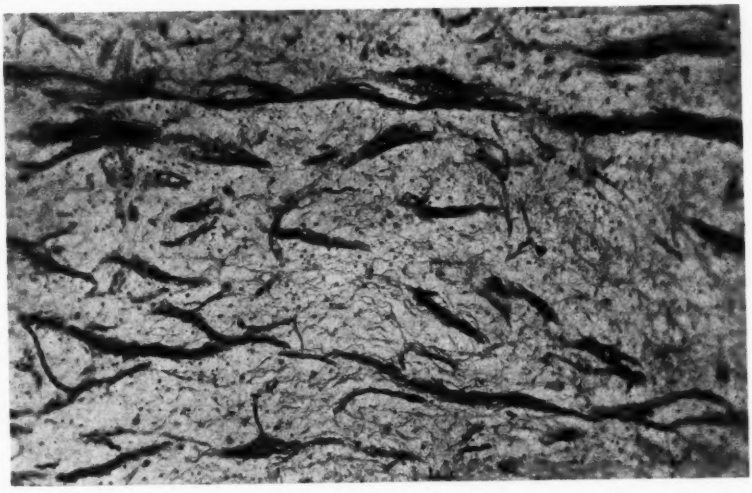

FIG. 2. Portion of nerve root showing wide separation of its components by the lipoma. Degeneration is present, but normal myelinated fibers are also seen. (Mahon stain)

Microscopic. The tumor was composed of an adult type adipose tissue interspersed by slender, slightly vascularized bands of fibrocollagen, containing also slender, irregular fascicles of peripheral nerve tissue (Figs. 1–4). With myelin stains, these showed occasional intact myelinated fibers, occasional fibers partially degenerated, and, in a few areas, nerve fascicles

in which neurolemmal proliferation was noted with a virtual absence of myelinated components. The cross-sections of some fascicles showed a few intact axons and scattered normal myelinated nerve fibers. In a few areas, slightly hyperplastic leptomeningeal tissue seemed to cover the fatty mass, and in other areas, fragments of dense fibrocollagenous tissue suggestive of dura were seen. Fat stains were strongly positive. There was no inflammatory change or old hemorrhage. In all sections there was a complete absence of any tissue that could conceivably have been derived from the spinal cord. Nissl and Cajal stains failed to reveal neurons or astrocytes. The neoplasm was therefore classified as of leptomeningeal origin, and while pathologically a lipoma, could conform with the characteristic behavior of a spinal meningioma of the lipoblastic type described by Cushing as "Type IX."

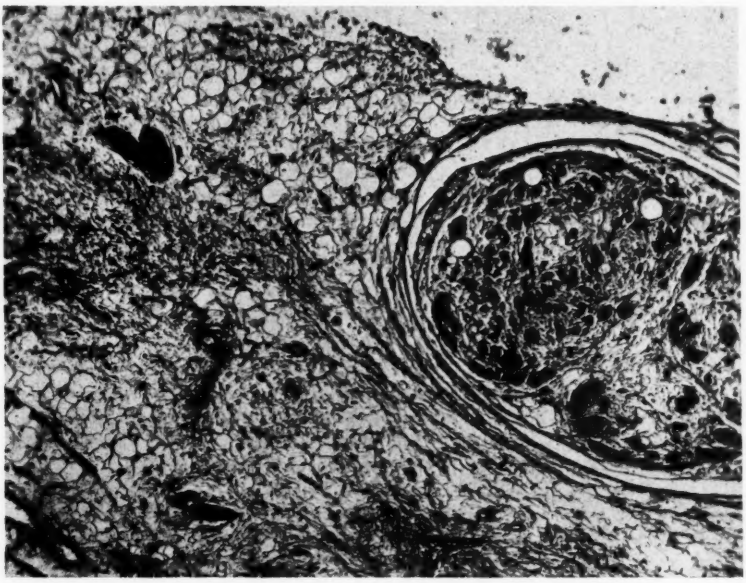

FIG. 3. View of tumor showing incorporation of a partially degenerated nerve root.

2nd Operation, Nov. 17, 1950. Because of the appearance of the biopsy material, further attempt at removal of the tumor seemed justified. The old wound was reopened. The cut edges of the dura were easily freed and retracted. The site of the previous incision into the tumor was now completely filled. A suture was put in the caudal portion of the tumor and gentle traction was applied in an attempt to rotate the spinal cord. With the traction, some demarcation appeared at the caudal end of the tumor. The pia mater was incised there and gentle blunt dissection freed a portion of tumor from the underlying spinal cord. The pial vessels coursing over the tumor were coagulated and gently dissected back to normal tissue. The tumor was then grasped and rotated to the left. Using blunt dissection, it was removed from its right lateral attachment to the spinal cord. It was necessary to cut through pia and a few dorsal root filaments which were incorporated in the left lateral margin of the tumor. Upon complete removal of the tumor it was seen that the spinal cord had been compressed into the right lateral portion of the spinal canal, stretching the left nerve roots. The tumor bed was rough and was not covered with pia. Two ruptured fascicles of the posterior columns of the spinal cord were seen. There was some capillary oozing in the margins of the tumor bed: this was easily controlled with thrombin-soaked Gelfoam®. The dura was closed with ease.

Pathological Examination. Four additional masses, measuring from 1 to 2.5 cm., were removed at the second operation. Microscopically the specimens from the first and second operations were identical.

Course. Postoperatively, the patient suffered a complete paraplegia, anesthesia below the 11th thoracic dermatome and complete loss of bladder control. Routine bladder catheterization was begun, but there was still dribbling, so that tidal drainage with an indwelling catheter was substituted. Sensation and motion began to return 2 weeks after operation. Flexor spasm

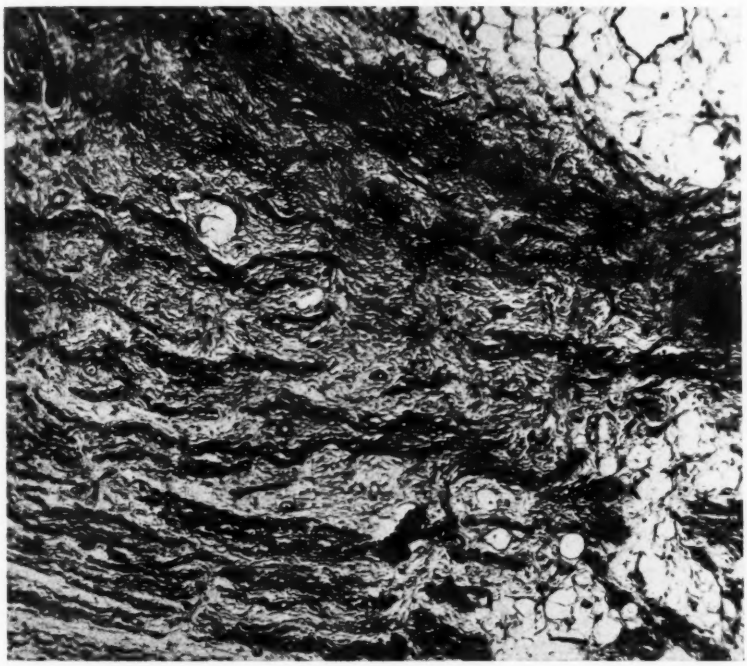

FIG. 4. View of displaced nerve root. There is infiltration of fascicles and fibrils by the adipose tissue of the neoplasm. (Hematoxylin and eosin stain)

finally disappeared during the 4th week postoperative, and intensive physical therapy was then begun.

By January, 1950, she had return of bowel and bladder control and was able to walk with assistance. In February, 1951, although weakness of the lower extremities was still present, she could walk unaided with knee-lock braces. There had been 75 per cent return of all forms of sensation except joint position and vibratory sense. These were completely absent. She stated that she occasionally had slight stress incontinence of urine.

In June, 1951, the patient reported that she was able to perform all her household duties and to care for her three small children without assistance.

DISCUSSION

This neoplasm arose from structures foreign to the spinal cord itself, being derived from meningeal components, apparently in the vicinity of the pia-arach-

noid. From histologic studies it appeared that the tumor arose at the junction of one or more nerve roots and the spinal cord. A reconstructed picture of the entire mass from the many fragments suggested a nerve root that had been distorted its fascicles being separated by ingrowing adipose and fibrous tissue which arose either at the junction between the nerve sheath and the leptomeninges or within the leptomeninges themselves. There was destruction of nerve roots by compression, as demonstrated by selective staining. The absence of tissue derived from the spinal cord in any of the many fragments examined confirmed the extramedullary origin of this tumor. The appearance of the apparently intramedullary growth at operation was compatible with that of a pressure displacement, particularly since the tumor was so intimately associated with nerve roots, close to their emergence from the cord.

This case is of interest because of the good surgical result that was obtained. Had not close attention been paid to the microscopic report of the biopsy taken at the first operation, the patient would have been given a hopeless prognosis. It was felt that because of the demonstrated benign nature of the neoplasm, a line of demarcation could be found between the lesion and normal spinal cord. Enucleation was possible only after the pia had been incised and the blood vessels on the surface of the spinal cord had been interrupted.

Although this report is not the place to discuss the histogenesis of intradural lipomas of the spinal cord, note should be made of the improbable intramedullary nature of these tumors. In the cases studied by Ehni and Love, 18 had sufficient description to determine the amount of medullary involvement by the neoplasm. Of these, 17 were undoubtedly pial or subpial, not in the substance of the cord. In some of these, the capsule of the tumor left a roughened bed on the denuded surface of the cord in the absence of the protecting pia mater. The subpial, but extramedullary location of these neoplasms has also been noted in the more recent reports.[2] It should be emphasized that when an intradural lipoma of the spinal cord is encountered, the pia mater should be incised in order to find a line of separation between the neoplasm and normal spinal cord.

SUMMARY

1. A case is reported giving detailed account of an intradural lipoma of the spinal cord.

2. Study of biopsy material resulted in a secondary operation and total resection of the neoplasm.

3. The extramedullary, but subpial location of these benign tumors is stressed.

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Obituary*

SIR HUGH CAIRNS

1896–1952

When people of the present writer's age die there may be regrets, but there is never the poignant feeling of unfulfilment that comes with the death of a younger man—shorn of the years in which to express himself fully and to deploy his gifts to

* Reprinted from the *British Medical Journal*, July 26, 1952, vol. ii, p. 233, with the kind permission of the Editor.

the utmost. The death of Sir Hugh Cairns is as sad a happening as could well be. It is not merely sad in the sense that we have lost from our midst a man whose company delighted us, but sad because British surgery has lost one of its most arresting figures so early, before he had given to the world what he had in store.

He had of course already accomplished so much that it is hard to know where to begin in writing about him, and in fact it would be possible to tell his story in several different ways, all of them correct. The young and handsome Australian from Adelaide, recently a Rhodes Scholar, who first made his mark at the London Hospital in the mid-nineteen-twenties had in 15 years become a national and almost as quickly an international figure. Hugh Cairns's influence was founded on the fact that he was supremely good at his work. He had a well-balanced and critical mind, but, critical though he was, he could be outstandingly generous in his appreciation of good qualities in others where he could perceive them. Thinking of him now and making this too-early judgment of him, one sees that it was remarkable that anyone should have combined all his gifts. I knew no one better as a surgeon, and scarcely anyone anywhere as good. I do not mean that he was brilliant to watch in the operating theatre, but that he gave the onlookers such confidence. As with Harvey Cushing, the utmost care had so obviously been taken in the working up of the case. Hugh Cairns demanded a very high level of performance from everyone associated with him. Some of his pupils have become leaders in neurosurgery in foreign lands.

Cairns prided himself on being "a good doctor." This lesson he had been taught by Harvey Cushing, who had it from Sir William Osler. Cushing used to say that you could tell whether a man was a good surgeon by noticing whether he personally saw that his patients were comfortable and was not too high-and-mighty to arrange their pillows. Hugh Cairns was fond of repeating this. It is not so small a thing as it sounds. It would be hard to say whether Harvey Cushing had a favourite above all others among his pupils, but certainly Cushing liked and admired no one more, recognizing the fibre in him. Cairns had a year in Boston as a Rockefeller Fellow in 1926–7, when he had already been appointed to the London Hospital. He had been a Rhodes Scholar at Balliol, a college that he loved, and where, to his pride, he was elected to a Fellowship in 1937. When he came back from the United States in 1927 he was resolved to devote himself entirely to neurosurgery, and, since no one had yet done that, many advised against it. However, his expert knowledge and his easy manners quickly triumphed.

He was well integrated as a person. I remember once making some remark about normality to Cairns, who replied that he thought that he himself was very normal. This estimate of himself was quite true. He was as normal a person as one could hope to know, so stable was he, so even tempered, so free from moods. But underneath he was a high-powered individual with immense ambitions to make his work and all about him as nearly perfect as he could. This he did by his own innate qualities and by collecting to him the best people he could find. This laid him open sometimes to criticism, for if he wanted something he usually got it. I never really found out whether he realized always that others saw easily through some of his schemes. He was not so simple as he thought he was. But he was simple in this, that he had a simple standard—perfection. Surely, he thought, no one can object to that. Nor can they. Rather of a piece was Hugh Cairns's estimate of his own ability. During the 25 years that we knew one other he said to me several times, "You know, I don't think I'm clever; I'm quite ordinary really." That again was true in a way. But Hugh Cairns's ordinariness was another man's excellence. He had in fact plenty of imagina-

tion, and he was creative. I suppose that what he meant by not being clever was not being fanciful. He was never that in his work, though he could be delightfully so in the family circle. He knew well enough his power to get things done, nor did he balk at unconventional approaches if they could attain ends: in that his Australian upbringing undoubtedly conditioned him, so that he was not prepared to accept customary channels if they seemed to him to be stupid or impeding. Nothing illustrates this better than the leading part he played in the formation of the new clinical school at Oxford in 1937. This was an exciting project, and Lord Nuffield's quick appreciation of the possibilities and his instant generosity made the dream come true almost as soon as it was dreamed. The Oxford clinical school was to be closely linked with the other university science departments, and a new and more scientifically trained doctor was to emerge. Somehow that was never quite achieved.

To the Army Cairns gave such services that they can never be over-praised. It is to the Army's credit that the leaders approved so readily the intelligence behind Hugh Cairns's unconventional approach to military planning. It was most appropriate that he should have returned to Australia and visited South Africa as Sims Professor in 1947–8. Occasions will surely arrive when his contribution to science, to our knowledge of the nervous system, to medical education, to his university of Oxford, to the creation of his superb unit at the Radcliffe Infirmary, and particularly to the wounded soldier can be told at proper length.

It is difficult to keep private grief out of a public message. But I and all of us cannot but most bitterly lament the departure of a character of such integrity, such power, such courage, and such charm. The next 10 years or so, his lost years, would have seen the real flowering of all that he had striven for so hard and so long. However, there is this to comfort us—men die, but those who have created something live on to influence other generations.

Illness unmasks a man, and in his last illness the true goodness and bravery of Hugh Cairns shone like a flame. His marriage to Barbara Smith, one of the remarkable children of a remarkable man, the then Master of Balliol, brought him such happiness as not everyone achieves. She and he were a beautifully matched pair. "Life is a pure flame," wrote Sir Thomas Browne, "and we live by an invisible sun within us." Hugh Cairns's sun has gone down, and it is dusk for more than him.

SIR GEOFFREY JEFFERSON

CORRESPONDENCE

OCULAR COMPLICATIONS OF CAROTID ANGIOGRAPHY

To the Editor:—In a paper written in collaboration with Doctor George W. Smith "The Ocular Complications of Carotid Angiography. The Ocular Signs of Thrombosis of the Internal Carotid Artery," (*J. Neurosurg.*, 1952, *9:* 517–537) I have made an erroneous statement as regards a portion of a paper written by Peter H. Schurr, F.R.C.S., "Angiography of the Normal Ophthalmic Artery and Choroidal Plexus of the Eye" (*Brit. J. Ophthal.*, 1951, *35:* 473–478).

On page 518 of our paper we stated: "According to Schurr the patient often describes a flash of light as the injection is being made. He has attributed this symptom, with which we have no experience, to retinal irritation." What was stated in the paper by Schurr is, "When diodone enters the circulation of the eye, it causes an intense burning pain for the duration of its stay there; this may rarely be accompanied by a flash of light."

Frank B. Walsh, M.D.

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Baltimore 1, Maryland

NOTICES

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASE

At the 32nd Annual Meeting of the Association for Research in Nervous and Mental Disease held in New York City on December 12–13, 1952, the following officers were elected for the year 1953: President, Dr. Davenport Hooker; 1st Vice-President, Dr. John C. Whitehorn; 2nd Vice-President, Dr. Lawrence H. Snyder; Secretary-Treasurer, Dr. Clarence C. Hare; Assistant Secretary, Dr. Rollo J. Masselink.

The subject for the 1953 meeting will be "Genetics and the Inheritance of Integrated Neurological and Psychiatric Patterns."

HARVEY CUSHING SOCIETY

The Program Committee for the annual meeting of the Harvey Cushing Society to be held in Hollywood, Florida, on April 22–25, 1953 is as follows: Dr. Joseph P. Evans, Cincinnati, Chairman; Dr. John F. Fulton, New Haven, and Dr. Franc D. Ingraham, Boston. Members who wish to present a paper should address their communications to Dr. Joseph P. Evans, Cincinnati General Hospital, Cincinnati 29, Ohio.

NEUROSURGICAL SOCIETY OF AMERICA

At the 5th Annual Meeting of the Neurosurgical Society of America held at The Cloister, Sea Island, Georgia, September 18–20, 1952, the following officers were elected: President, Dr. William F. Meacham, Nashville; Vice-Presidents, Drs. Kenneth E. Livingston, Portland and Harry P. Maxwell, Milwaukee; Treasurer, Dr. I. Joshua Speigel, Chicago; Secretary, Dr. C. D. Hawkes, Memphis.

SOCIETY OF NEUROLOGICAL SURGEONS

The next meeting of the Society of Neurological Surgeons will be held in New Orleans, Louisiana on March 19–22, 1953, with Dr. Dean Echols as the official host. Attendance is for the membership and the invited guests.

THE COMPLICATIONS OF CAROTID ARTERY LIGATION IN THE NECK

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(Received for publication July 25, 1952)

A MAJOR definitive method in the treatment of intracranial aneurysms and other vascular abnormalities is ligation of the carotid artery or one of the branches in the neck. Survey of the literature revealed that there are no data as to the efficacy of this method and that it is frequently followed by serious sequelae. Summaries of this literature are given in the publications of Pilcher and Thuss[17] and Dandy.[4] It is the purpose of this paper to discuss the etiology, prevention, and treatment of such complications in the light of 65 consecutive cases of carotid ligation in the neck.

MATERIAL

All cases of carotid ligation in the neck performed for intracranial vascular abnormalities at the Presbyterian Hospital and Neurological Institute during the years 1943–1950 were surveyed. Lesions were classified as unverified unless precise location by arteriogram or operation was known. It is realized that the number of cases in the present report is too small to be of statistical significance and percentages are given only to facilitate presentation of the material.

Of the 65 cases, there were 21 instances of cerebral complications and 6 deaths, giving a case morbidity of 32 per cent and a case mortality of 9 per cent. Complications ranged from appearance of pathological reflexes to hemiplegia and other severe disturbances. Two deaths occurred in terminal patients and may not have been related to the ligation; they are included, however.

Table 1 summarizes the cases according to location and type of lesion, with incidence of complications and deaths. It indicates that the majority of these complications occur when the lesion lies on or near the circle of Willis.

FACTORS IN THE PRODUCTION OF COMPLICATIONS

Age. Fig. 1 indicates that the incidence of complications does not increase with advancing age. These data are in accordance with those of Pilcher and Thuss[17] and Schorstein.[20] Furthermore, the average age according to the various locations of the aneurysms did not vary significantly so as to weight the latter figures unduly (Table 2). Thus in this series age per se did not play a role in the production of complications.

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TABLE 1

Location of lesion, with incidence of complications and deaths

Location of Aneurysm	Number of Cases	Complications		Deaths	
		No.	%	No.	%
Internal carotid					
A-V in cavernous sinus	7	1	14	0	
Infraclinoid carotid	11	2	18	0	
Supraclinoid carotid	12	6	50	2	16
Anterior cerebral	4	0		0	
Middle cerebral	2	0		0	
Posterior cerebral	0				
Anterior communicating	3	2	66	0	
Posterior communicating	9	6	66	1	11
Arteriovenous malformations	7	0		1	14
Unverified	10	4	40	2	20
Totals	65	21	32	6	9.2

Bleeding. Survey of the literature shows that the mortality for a given bleeding episode ranges from 11 per cent to 60 per cent. The efficacy of carotid ligation in the neck in arresting hemorrhage cannot be learned since no differentiation between active and arrested bleeding has been made. When 10 days to 2 weeks have elapsed without further bleeding, it may be assumed that spontaneous arrest has occurred and ligation done after this time does not arrest hemorrhage but can only function to possibly prevent further bleeding episodes in the future.

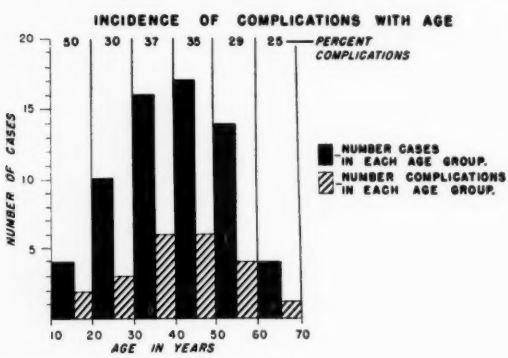

FIG. 1

In Table 3, 19 patients with bloody spinal fluid are assembled in 2 groups: (A) those ligated within 10 days of bleeding and (B) those ligated

TABLE 2

Average age of patients according to various locations of aneurysms

Locations	Internal	Anterior	Middle	Ant. Comm.	Post. Comm.	Malform.
No complication	45 yrs.	39 yrs.	50 yrs.	39 yrs.	42 yrs.	21 yrs.
Complication	36 yrs.			40 yrs.	39 yrs.	

more than 10 days from bleeding. The higher morbidity and mortality in the first group may be noted. Two additional facts are of interest. Two of the 3 complications in the first group and 5 of 6 complications in the second group were associated with lesions lying astride of the circle of Willis, chiefly the communicating arteries. In Group B it is of interest to note the number of patients who have done well 2 to 5 weeks before elective ligation

TABLE 3

Relation of complications and death to fresh subarachnoid bleeding

Case	Interval (days)	Number of Hemorrhages	CSF Pressure	Complication	Death
		Group A—Fresh Bleeding			
V.P.	0	2	160 mm. H_2O	+	+
E.P.	0	1		+	0
M.McH.	0	2	300	0	+
M.R.	4	4	210	0	0
A.S.	5	4	140	+	+
Totals				3 (60%)	3 (60%)
		Group B—Arrested Bleeding			
A.B.	14	2	280 mm.	0	0
P.H.	15	2	180	0	0
W.G.	20	1	390	+	0
L.H.	20	1	240	0	0
A.S.	20	1	220	0	0
R.K.	20	1	260	0	0
F.M.	22	1	270	0	0
R.C.	28	3	130	0	0
L.H.	36	3	200	+	0
R.S.	40	1	380	+	0
H.H.	43	1	250	+	0
W.S.	46	2	360	+	0
S.D.	55	3	280	0	0
V.F.	74	1	570	+	+
Totals				6 (43%)	1 (7%)

was done. This raises some question that ligation must be done within 2 weeks and also casts some doubt on the role that ligation does play in the prevention of recurrent hemorrhage.

Other authors[6,12,13,20] have emphasized the high morbidity and mortality accompanying ligation in the face of fresh subarachnoid bleeding, especially when associated with aneurysms lying on or near the circle of Willis. Falconer[9] presented a more optimistic report of 39 cases of leaking aneurysms, of which 24 were ligated in the neck with a mortality of 13 per cent. However, times from bleeding to ligation were not given and only 5 of the lesions were in association with the circle of Willis.

This series, plus reports from the literature, suggest that there is a definite

hazard to ligation of the carotid in the neck in the face of active bleeding and that location of the lesion astride the circle of Willis imposes an equal or greater hazard. In addition, ligation may not effectively control bleeding. A level of 50 mm. Hg arterial pressure is still twice the usual (300 mm. H_2O) CSF pressure, so that bleeding may continue. However, pressure in the terminal cortical circulation, physiologically at lower levels, may be so reduced by ligation that it cannot exceed the increased intracranial pressure and anemic sequelae result. We therefore believe that ligation, when indicated, is best deferred for 2 weeks from date of the last bleeding episode.

Previous Disease. The low incidence of complications after the age of 50 makes it unlikely that arteriosclerosis per se is a factor in complications. Likewise, there was no difference in rates between the hypertensive and normotensive patients in the series. Previous studies[22] have shown that both groups exhibit the same drop in pressure percentagewise.

The Matas Test. It has been frequently stated that a negative Matas test precluded complications from carotid artery ligation. Recently this has been shown not to be true,[1,22] and the experience in the present series is in agreement with this. All complication cases were associated with negative Matas tests ranging from 10 to 30 minutes. Furthermore the period between ligation and onset of complication constituted a further test under most positive conditions of occlusion without giving warning of danger. Hence extension of the test beyond the 30-minute period is not indicated, especially since it can be shown by pressure recordings that it is impossible to maintain complete occlusion for more than a few minutes. While the negative test is not contributory, testing should always be carried out to discover those patients who present a positive test which may contraindicate ligation.

Pressure Levels Following Ligation. It is believed at present that extremely low pressures following ligation are indicative of a deficient collateral circulation. Table 4, presenting data on those cases in which pressure studies were done, indicates that absolute or percentage drops in pressure levels following ligation may not be a reliable guide as to whether complica-

TABLE 4

Correlation of complications with intracarotid pressure levels after ligation

Case	Location	Artery Ligated	Type	Pressures		Complication
				Before	After	
L.H.	Ant. communicating	Common carotid	Complete	130/80	54/40	+
S.D.	Ant. communicating	Common carotid	Partial	120/80	100/80	0
R.D.	Internal carotid	Common carotid	Complete	150/80	60/40	0
M.R.	Middle cerebral	Common carotid	Complete	120/80	30/27	0
C.T.	AV angioma	Internal carotid	Complete	130/80	43/33	0

tions will ensue. This is because factors other than pressure alone undoubtedly play a role.

The Nature of the Ligation. The various aspects of the surgical procedure itself were examined to see what role they might play in the production of complications. With regard to anesthesia, there was no difference in the incidence of complications whether done with avertin, pentothal, or local procaine with infiltration of the carotid sinus. Therefore it is believed that local procaine is the anesthesia of choice since the patient can be readily examined after ligation.

The type of ligature material used (braided silk, linen tape, aluminum or tantalum bands) made no difference in the occurrence of complications. We therefore have favored 1 cm. tantalum bands to approximate a broad band of intima under least traumatic conditions and to facilitate removal

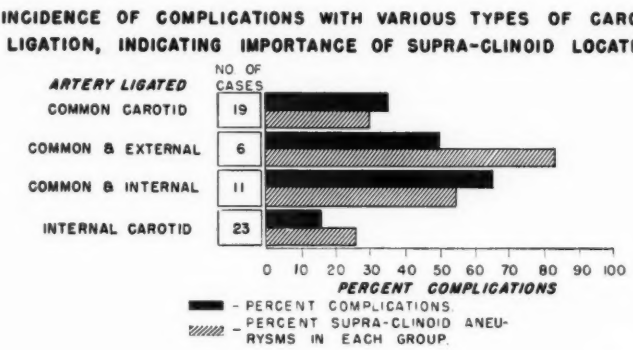

FIG. 2

should this be necessary. The recommendation of Rogers[19] for ligation and division of the artery to avoid thrombosis and embolism cannot be accepted since the latter are not responsible for the usual type of complication. Should complications develop, there is no way to restore the circulation if the artery has been divided.

Turning to a consideration of ligation of the common carotid and each of its branches, it was found that the lowest incidence of complications was associated with ligation of the internal carotid artery (Fig. 2). This was unexpected and the question immediately arose as to whether the number of supraclinoid (supraclinoid internal carotid plus the communicating arteries) aneurysms varied significantly one group from the other so as to unduly influence the figures. Fig. 2 shows that in a general way the incidence of complications varies in accordance with the percentage of supraclinoid aneurysms in each group.

These supraclinoid aneurysms were then segregated and the incidence of complications for each type of arterial ligation was compared (Fig. 3).

The very high incidence of complications for the group as a whole is noted. When, on the other hand, one excludes these supraclinoid lesions from consideration and studies the incidence of complications with the various

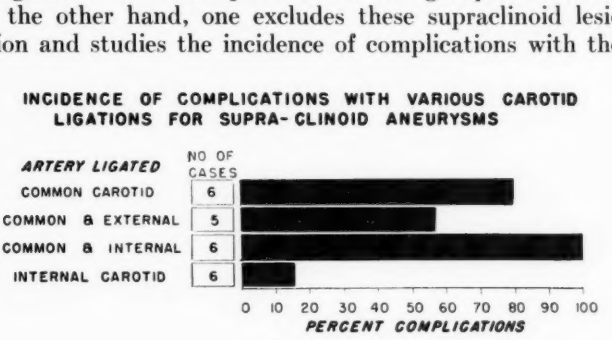

FIG. 3

types of ligation done for infraclinoid aneurysms (Fig. 4), one sees that the risk is low and essentially the same no matter which artery is ligated. Reference to Figs. 3 and 4 shows that the low incidence of complications with internal carotid artery ligation in Fig. 2 is because of the large number of infraclinoid aneurysms exposed to this type of ligation.

These data suggest that it is the location of the lesion with reference to

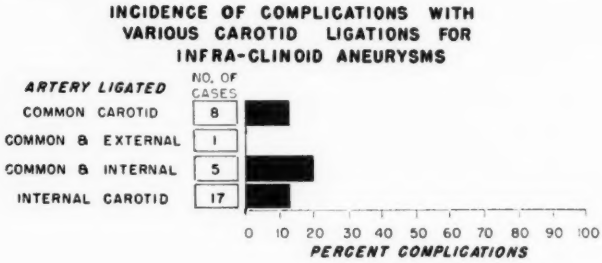

FIG. 4

the circle of Willis and the collateral circulation rather than the particular artery ligated that determines the risk from a given ligation. At the present time no one type of ligation can be recommended over another for these reasons.

Further tabular analysis* shows that ligation in stages and various other combinations of ligations, whether in one or two stages, do not affect the incidence of complications. The reasons for this have been clearly shown by arterial pressure recordings. An example is seen in Fig. 5 in which more than 75 per cent occlusion of the artery failed to produce any change in pressure. As a first stage in a stage ligation it would be without effect,

* Omitted because of limitation of space but available from the author on request.

either in reducing pressure or inducing collateral circulation. There is further no evidence that partial ligations with irritants such as cellophane ever produce a later complete occlusion and hence cannot be regarded as ligations. In regard to the procedure of common carotid ligation followed in time by external or internal ligation, it has been shown[1,22] that the additional ligation may raise, lower, or have no effect whatever on the resulting

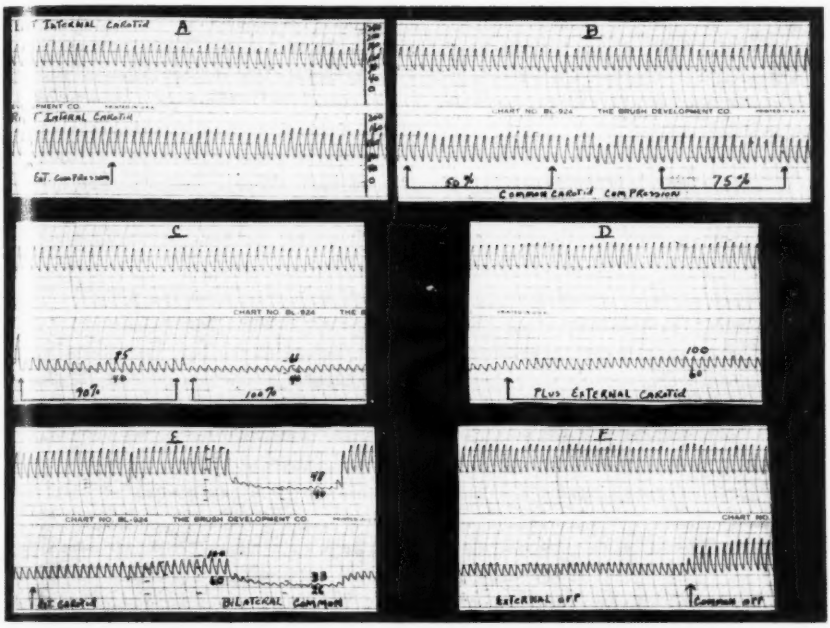

FIG. 5. Relation of partial ligation to pressure reduction. Catheters in both internal carotid arteries, the right carotid system being exposed. (A) Compression of right external carotid—no change. (B) 50% and 75% occlusion of right common carotid—no change in pressure. (C) 90% occlusion of common carotid—50% reduction in pressure. Complete occlusion gives pressure of 61/40 mm. Hg. (D) Additional occlusion of external carotid causes *rise* in pressure to 100/60 mm. Hg. (E) Bilateral carotid occlusion reduces pressure to 33/26, indicating possibly deficient collateral circulation.

arterial pressure. Thus in some cases, common carotid ligation causes greater reduction in pressure than does internal carotid ligation. For these reasons stage ligation cannot be recommended.

Reports in the literature[4,5,9,14,19] advocating one or another type of carotid artery ligation are thus difficult to evaluate in the light of the above considerations. A consideration of the present data indicates that it is the location of the lesion and not the artery ligated that determines the incidence of complications.

Type of Lesion. Table 1 shows that the incidence of complications from carotid ligation is materially lower in those cases in which an arteriovenous

communication exists, either from aneurysmal rupture into the cavernous sinus or in arteriovenous malformations of the hemisphere. In one case all the carotid branches were ligated bilaterally for a parietal angioma, without sequela. In a case of arteriovenous malformation of the temporal lobe, common carotid ligation produced a pressure of 27/25 mm. Hg in the distal internal carotid; this was well tolerated. Shenkin *et al.*[21] have demonstrated a 3-fold increase in cerebral blood flow in these lesions. In addition, it should be noted that these lesions are located away from the circle of Willis and do not interfere with potential collateral circulation. Although it thus appears that this group offers less risk to ligation, the less favorable report of Olivecrona[15] is noted, emphasizing the hazards of ligation and its doubtful therapeutic value.

Location of the Lesion and the Collateral Circulation. As has been intimated previously, these two considerations are of the utmost importance in the etiology of complications following ligation of the carotid artery in the neck. The importance of the location of the lesion can be seen by reference to Table 1 and Figs. 2, 3 and 4. Although the numbers are small, the strikingly higher incidence of complications with supraclinoid internal carotid and, in particular, communicating artery aneurysms is significant. Thus 37 per cent of the aneurysms accounted for 66 per cent of the complications. These aneurysms, lying astride of the collateral branches of the circle of Willis, are in a vital position as regards the production of complications. A vessel whose patency is maintained by normal arterial pressure may, under contiguous pressure of an aneurysm, collapse following reduction of pressure secondary to carotid ligation.[20] In addition, it has been emphasized that the circle of Willis is more frequently deficient in the presence of an aneurysm.[16] These considerations have indicated the necessity for complete arteriography, complemented if possible by pressure recordings, in order to check the competency of the collateral circulation.[1] It has been stated[9] that strong collateral circulation negates the effect of carotid ligation but it is submitted that without it ligation cannot be tolerated.

In 16 of the 21 complication cases arteriography was carried out. In the 3 cases in which contralateral compression was done, the anterior circulation was deficient in 2 and the posterior in the other. In an additional 3 cases, deficiencies of the communicating arteries were present although contralateral compression was not done. In 1 case the middle cerebral failed to fill because of pressure of the aneurysm. In the remainder of the cases, no conclusion could be drawn relative to the collateral circulation. These meager data have indicated to us the necessity for complete arteriographic visualization of the collateral circulation prior to ligation.

THE CLINICAL SYNDROME

Routine testing of patients at 15-minute intervals for the first 12 to 24 hours after ligation afforded opportunity for detailed study of their clinical course. The more important features of the cases in which complications developed are summarized in Table 5.

Seventeen of the complications occurred within 12 hours; 4 after 18 hours. The onset was sudden in a period of minutes after the last normal observation.

In a careful study of the vital signs after ligation, it was found that in 9 of the 21 patients with complications there developed a significant rise in arterial pressure. This hypertension bore no relation to the artery ligated nor did electrocardiograms when done show any evidence of carotid sinus effect. While 43 per cent of the patients with complications showed hypertension after ligation, only 9 per cent of those who had no complications showed such a rise. Thus hypertension at any time after ligation may warn of impending danger. In the 3 complication cases in which the spinal fluid pressure was measured, it was elevated.

Of the 21 patients with complications, 13 complained of headache, mental confusion occurred in 7, hemiparesis in 17, hemisensory changes in 6, aphasia was present in 6 out of 17 with left-sided lesions, and blindness occurred in 2. It is of interest that only 3 patients were unconscious, 2 of whom were in coma from bleeding when ligation was performed.

In Table 5 it will be noted that in 10 cases the ligature was not removed following the onset of complications. Study of these cases shows that the 5 patients with most minimal symptoms (asterisk in "Time of Clip Removal" column) made spontaneous recoveries; the other 5 showed slight if any improvement, and 2 died. The 11 cases in which clip removal was done presented more profound types of complications. In these cases timely removal of the clip was lifesaving, as discussed below (Treatment).

It is very unlikely that spontaneous recovery would have ensued. The speed and degree of recovery was directly correlated with the promptness with which the clip was removed, 15 minutes being the limit which could not be safely exceeded.

ETIOLOGY

A number of recent reviews have presented the various theories of the causation of complications after carotid artery ligation in the neck.[4,5,13,15,20] The following considerations regarding these theories seem pertinent at this time.

1. *Hemorrhage.* Although in a few cases persistent bleeding despite ligation is the sole cause of complications, in others ligation in the presence of increased intracranial pressure results in encephalomalacia for reasons already mentioned. In only 2 cases (V.P. and A.S.) in the present series was it likely that continued bleeding may have been responsible for death. It appears that there should seldom be doubt as to whether bleeding is occurring and that hemorrhage is not a factor in the production of the majority of complications save where it raises the intracranial pressure and thus so compromises the terminal circulation that cerebral ischemia follows ligation.

2. *Thrombosis.* In no case in the present series could thrombosis or embolism be established a causative agent, although in several instances

TABLE 5

Summary of cases in which complications developed after carotid artery ligation

Case	Location	Preoperative Symptoms and Signs	Artery Ligated	Onset	Vital Signs
E.P.	Int. C.	Bilat. loss vision, diplopia.	C.C.	Immed.	Normal
H.H.	Cavern. AV fistula	Proptosis, chemosis, bruit. 95% shunt by arteriogram.	Int. C.	Immed.	Normal
V.P.	Supraclin. C.	2 days bleeding, hemiparesis. Rt. V, VII.	C.C.	Immed.	Normal
W.R.	Supraclin. C.	2 yrs. loss smell, vision, libido. Polyuria, central scotomata.	C.C.+int. C.	Immed.	↑ P&BP
A.F.	?infraclin.	2 yrs. diplopia, ptosis, facial pain. III, IV, V (1 & 2)	C.C. in stages	Immed.	↑ P&BP
H.W.	?infraclin.	2 yrs. diplopia, ptosis. Rt. III, VI.	C.C. 50%	30 min.	↑ BP
C.H.	Post. commun.	Hypertension; pain & ptosis rt. eye. Rt. III, IV, VI.	C.C.	23 min.	Normal
N.M.	Ant. commun.	2 bleeding episodes, memory loss. Rt. hyper-reflexia.	C.C.+ext. C.	40 min.	↑ P&BP
L.H.	Ant. commun.	6 days: 3 bleeding episodes.	C.C.	2 hrs.	↑ P&BP
A.S.	?infra C.	6 yrs. intermittent diplopia, rt. hemiparesis. 4 bleeding episodes, coma.	Int. C. in stages	5 hrs.	Normal
A.C.	Post. commun.	2 bleeding episodes. Rt. III.	C.C.+ext. C.	6 hrs.	↑ BP
W.S.	Supraclin. C.	2 bleeding episodes. Decreased vision.	C.C.+int. C.	7 hrs.	Normal
R.S.	Post. commun.	10 yrs. ptosis. 3 hrs. bleeding, seizures, rt. hemiparesis.	C.C.+int. C.	8 hrs.	↑ P&BP
V.F.	?infra C.	Hypertension. 6 hrs. ptosis. Lt. VI.	Int. C. 60%	9 hrs.	↑ BP
A.Z.	Supraclin. C.	Facial pain, ptosis, diplopia. Lt. III, V (1, 2, 3).	Int. C.	10 hrs.	Normal
G.J.	Post. commun.	Diplopia, blurred vision, ptosis. Lt. III, VI.	C.C.+int. C.	12 hrs.	↑ P&BP
M.M.	Supraclin. C.	1 yr. facial pain, diplopia, ptosis. Rt. III, IV.	C.C.+int. C.	13 hrs.	Normal
W.G.	Post. commun.	3 days pain rt. eye, ptosis, bleeding. Rt. III, VI.	C.C.+ext. C.	18 hrs.	Normal
H.H.	Post. commun.	Ptosis, bleeding. III, IV, V (1), VI.	C.C.+int. C.	18 hrs.	↓P&BP
C.H.	Infraclin. C.	Diplopia, facial pain and numbness. Bruit; V, VI.	C.C. in stages	58 hrs.	Normal
H.P.	Infraclin. C.	1 yr. decreased vision. Optic atrophy, lt. homon. hemianopia.	Int. C. in stages	72 hrs.	Normal

TABLE 5—(*Continued*)

Case	Signs of Complication	Time of Clip Removal	Artery	Treatment	Result
E.P.	Operative rupture aneurysm. PO hemiparesis.	0		Supportive	Stormy course 2 mos. Residual facial.
H.H.	Blindness ipsilat. eye.	0*		0	Spontaneous recovery 3 hrs.
V.P.	Complete hemiplegia, decerebrate.	0		0	Died 1 day PO.
W.R.	Marked PO mental confusion, disorientation.	0		0	Discharge 6 days; still profane, irritable, abusive.
A.F.	Lt. Babinski, unconscious.	Immed.	Patent and pulsating	0	Immed. recovery. 35% occlusion well tolerated.
H.W.	Sl. lt. hemiparesis, bilat. Babinski.	Immed.	Patent and pulsating	0	Immed. recovery. 3-stage C.C. ligation successful.
C.H.	Facial weakness, bilat. Babinski.	1 min.	Patent and pulsating	0	Complete recovery 30 min.
N.M.	Confusion, aphasia, sl. weakness rt. arm and face. Drowsy.	0*		Caffeine, CO_2 papaverine	Cut sutures to remove clip. Awakened. Signs gone.
L.H.	Hemiparesis, confusion, bilat. blindness. Pupils react well.	5 min.	Patent and pulsating	CO_2, arterial heparin, papaverine	Hemiparesis gone 30 min. Vision normal 42 hrs.
A.S.	Gradual aphasia, Babinski, flaccid hemiplegia.	0		0	Deepening coma. Death 2 days PO. ? bleeding.
A.C.	Nausea, vomiting, lt. Hoffmann.	0*		0	Spontaneous recovery.
W.S.	Lt. hemiparesis in 5 min. period.	20 min.	Patent and pulsating	0	Partial recovery 1 hr.; complete in 6 hrs.
R.S.	Aphasia, complete hemiplegia, unconscious.	15 min.	Int. C. patent	Arterial heparin, CO_2, stellate block	Immed. 25% recovery. 90% 2 wks.; residual.
V.F.	Bed high gatch. Vomit, aphasia, rt. hemiplegia and hyperesthesia.	30 min	Patent and pulsating	0	Sl. immed. improvement, relapse 8 hrs.; died 20 days PO.
A.Z.	Vomit, apprehensive, aphasia, rt. hemiparesis.	20–35 min.		0	No improvement. Died 2 days PO.
G.J.	Hemiplegia, hemianesthesia, aphasia, Babinski.	20 min.	Int. C. patent	CO_2, heparin, stellate block, papaverine	No improvement. Died 20 days PO.
M.M.	Restless, sudden lt. flaccid hemiplegia, hemianesthesia.	30 min.	Patent and pulsating	0	Partial improvement; residual weakness arm and face.
W.G.	Very sl. weakness lt. arm and face.	90 min.	Patent and pulsating	Papaverine, heparin	Immed. recovery after clip removed.
H.H.	Sl. emotional facial weakness after C.C. ligation.	0*	Normal at 2nd op.	Ephedrine	Cleared after 2 days.
C.H.	Numbness and 25% weakness lt. hand.	0*		0	Spontaneous recovery in 3 hrs.
H.P.	Confusion, lt. hemiparesis, marked facial weakness.	0		0	Sl. improvement. Facial weakness and hemiparesis after 4 yrs.

*=spontaneous recovery.

they could not be excluded. In 11 cases of two-stage ligation, the common carotid ligated at the first stage was subsequently found to be thrombosed for some distance distal to the clip; in no case did complication result from this thrombosis. In all 10 cases in which clip removal was done the artery was found to be patent prior to clip removal and pulsated freely following removal. Recovery paralleled time required for clip removal. Even when symptoms appeared at 18 hours, in 1 case they cleared immediately upon release of the clip, and in the other, the artery was found to be normal at a second operation. When symptoms appeared at 58 hours, spontaneous recovery over a 3-hour period suggested a period of ischemia. Schorstein[20] reported a 5-day progressive hemiplegia with death on the 13th day; at autopsy the carotid tree was clean and a typical anemic infarction was present. In a case more recently observed than the present series, and to be reported elsewhere, aphasia and hemiplegia appeared suddenly at 57 hours after ligation; prompt clip removal resulted in complete recovery within 30 minutes.

Occasionally, extension of thrombus from the aneurysm itself may cause cerebral sequelae[7] but in the main, few documented reports of thrombosis as the cause of complications from ligation exist. The experience of the present series and review of the literature indicate that cerebral ischemia is of much greater significance. For these reasons, the techniques of double ligation and division, and of partial ligation, both done to avoid the complication of thrombosis, are not indicated, since the former precludes the possibility of recovery when the more common cerebral ischemia ensues, and the latter is not really a ligation.

3. *Vasoconstriction.* There is no evidence to indicate that constriction of the cerebral vessels follows ligation in the neck. Cerebral vessels constrict significantly in response to mechanical stimuli,[10] and high oxygen and low CO_2 concentrations; none of these factors obtains following ligation. Response to sympathetic stimulation is insignificant.[8,11,18]

There is strong presumptive evidence that vasodilatation follows carotid ligation. Lowered oxygen tension, elevated CO_2 tension, and lowered blood pressure all follow ligation; they are all very potent dilators of the cerebral vessels. Further, ligation is known to cause dilatation in the tissues distal to the ligation. Finally, increased concentration of metabolites secondary to the slowed circulation produces vasodilatation. Thus of the factors reasonably expected to be present following ligation, all are vasodilators and none are vasoconstrictors. This may explain the disappointing results from the use of dilating drugs and stellate ganglion block.

In a number of clip removals, it is the rule to see the artery distal to the clip collapsed to very small caliber, suggesting spasm. The artery has always regained its normal caliber and pulsation upon removal of the clip. The narrowed diameter is thus the expression of an apulsatile pressure inadequate to fill the vessel normally.

As previously noted, EKG and pressure recordings revealed no evidence of carotid sinus effects.

4. *Cerebral Ischemia.* As indicated in previous sections, it is believed that this is the process responsible for the majority of complications from carotid ligation in the neck. The crucial role of the collateral circulation, the dangers of lowered arterial pressure by ligation in the presence of increased intracranial pressure, and the recovery that ensues with timely clip removal have all been discussed. The following tentative hypothesis is suggested for the changes that occur. Insufficient blood flow secondary to ligation and deficient collateral circulation cause a relative anoxemia. This produces anoxia of the terminal vessels with dilatation, transudation of fluid, unilateral cerebral edema, and increased intracranial pressure. Compensatory arterial hypertension ensues but its effect is blocked by the ligation. As the process continues, tissue anoxia follows until the compensation point is passed, when clinical symptoms make their rapid appearance, the nature and degree depending on the location and degree of the anoxic process. This process may be reversed by prompt removal of the ligature with re-establishment of adequate blood flow and oxygenation. Delay in clip removal produces results similar to delay in resuscitation following cardiac arrest.

While the data are admittedly incomplete, this hypothesis gives the present advantage of providing what has been the basis for active and, in more recent cases, successful prevention and therapy for these distressing complications.

PATHOLOGY

Schorstein[20] has summarized the available data on the pathological changes with this type of cerebral complication from carotid ligation. The 1 case in our series in which autopsy was performed is reported below.

CASE REPORT

G.J. was a 30-year-old female admitted in July 1949 with history of left-sided headaches, ptosis, blurred vision, and diplopia, and without a history of bleeding.

Examination. There was paralysis of the left 3rd and 6th nerves. There was no bruit. Arteriography confirmed the presence of a left posterior communicating saccular aneurysm but since contralateral compression was not carried out, no estimate of the collateral circulation was obtained. The Matas test was negative after 30 minutes. Vital signs and CSF pressures were normal, the fluid being clear.

Operations. On July 16, 1949 the left common carotid was occluded by a 1 cm. tantalum band without incident. On July 23, 1949 the internal carotid was occluded without difficulty by a similar band.

Course. The patient was tested every 15 minutes and was last found to be normal at 5:30 A.M. At 5:45 A.M., 12 hours after ligation, she was found to be hemiplegic, hemianesthetic and aphasic, but conscious and alert. The systemic arterial pressure was 190/120. The CSF pressure was 220 mm. H_2O and the fluid was clear. The clip was removed at 6:05, within 20 to 35 minutes, and a stellate block was done. The artery was patent and pulsating following clip removal. Heparin, papaverine, nicotinic acid, O_2 and 7 per cent CO_2 were given. The patient showed little improvement and died on the 15th postoperative day.

Autopsy. The common carotid artery was thrombosed from its clip to the carotid

bifurcation in the neck (proximal to band on internal carotid) but the internal carotid and all its branches were free of thrombi and emboli. There was no evidence of damage to the wall of the internal carotid artery at the site of the previous clip. Thus careful examination of the internal carotid vascular tree failed to reveal anything that could conceivably have caused this complication. Examination of the brain revealed an anemic infarct of the left frontal and parietal lobes in the distribution of the left anterior and middle cerebral arteries. The frontal lobe was atrophied and there was swelling of the parietal lobe, with herniation of the cingulate gyrus and uncus.

Microscopic Examination. There was endothelial hyperplasia of small vessels limited to the distribution of the middle and anterior cerebral arteries. Typical changes of ischemic infarction were present in the cortical grey and subcortical white matter, as well as in the globus pallidus and anterior limb of the internal capsule. These changes consisted of virtual absence of neuronal elements, edema of the tissue, increase in astrocytes and mesodermal elements, and dilatation of the finer vessels. These changes fulfill the criteria for anoxic changes in the brain as presented by Courville.[3]

These findings are in all essentials similar to those reported by Schorstein.

TREATMENT

In the light of previous considerations, the following is suggested as an outline of treatment of these complications.

1. Adequate pre-operative knowledge of the dangerous cases, with emphasis on the supraclinoid and communicating artery location and adequacy of the collateral circulation.

2. Selection of the optimum type of ligation aided by complete arteriography and pressure recordings.

3. Adequate observation and testing during the first 24 hours. Since the clip must be removed within 15 minutes and 10 minutes is usually required for such removal, we have found that the patient must be under constant surveillance by a nurse and tested for strength and responsiveness every 5 minutes for at least 12 hours.

4. Adequate facilities for clip removal are kept on a sterile tray by the bedside and a doctor is instantly available to carry out removal in bed and without anesthesia if necessary. As in cardiac arrest cases, time is of the essence and must be carefully noted.

5. Supportive therapy, especially high O_2 atmosphere, and careful attention to the airway are necessary. Tracheotomy should be promptly carried out when indicated, since hypoxia cannot be tolerated by these patients recovering from an episode of critical cerebral anoxia. The use of accessory muscles of respiration, pulse and blood pressure changes, and cyanosis are late signs of hypoxia and cannot be relied on.[2]

6. To offset the possibility of sludging, intra-arterial heparin may be given through a fine catheter.[1] The efficacy of this procedure is not known at present. The use of vasodilators, such as nicotinic acid, papaverine,

stellate block, and 7 per cent CO_2, has been disappointing, since the vessels are probably maximally dilated from anoxia secondary to the ligation.

SUMMARY AND CONCLUSIONS

A review of 65 consecutive cases of ligation of the carotid artery in the neck for various intracranial vascular lesions has been carried out with a view to establishing, if possible, those factors that may contribute to the production of complications from this procedure. The importance of the supraclinoid location of the lesion in the vicinity of the circle of Willis and the necessity for adequate collateral circulation have been emphasized. The typical clinical picture and some brief considerations as to etiology of the complications have been presented, with emphasis on ischemic anoxia as the predominant etiologic process. On the basis of this experience, some suggestions have been made as to avoidance and treatment of these complications as they arise, stressing the need for close observation during the postoperative period and prompt removal of the clip or ligature when complications ensue.

Because it is realized that the series is small, it is not possible to draw definite conclusions on general or statistical grounds until further data are available. It is hoped that the report may be of some assistance to those interested in the problem and provide a basis for more active therapy when such complications arise.

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SYMPATHETIC BLOCKADE, AN ACUTE CERVICAL CORD SYNDROME

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IN 1912 it was observed that spinal or decerebrate animal preparations were unable to maintain normal body temperature in response to change in environmental temperatures.[5] The critical level of section was found to be in the region of the optic thalamus, and transections made distal to this juncture produced poikilothermia. Irregularities in body temperature of humans following severe spinal cord injuries have been seen. The usually described trend is hyperthermia because of the absence of sweating peripheral to the lesion. Changes in cardiac rate have also been noted and have been considered a dire prognostic sign.[1,4] It is the purpose of this communication to delineate a rather frequently observed syndrome in war injuries of the cervical spine, to discuss the neurophysiological principles involved and to outline a method of treatment.

In a series of spinal cord injuries, caused by penetrating wounds or severe flexion trauma, treated in mobile and fixed neurosurgical installations in the Korean campaign during a 22-month period from September 1950 to July 1952,† 321 laminectomies were done, 46 of them operations on the cervical spine (14.3 per cent). The overwhelming majority of the injuries were caused by shell-fragment penetrating wounds of the spinal canal. In 26 of the 46 cases of cervical cord injuries that were operated upon, all or a part of an acute cervical syndrome was present.[6] The syndrome, which suggests an "acute sympathetic blockade," is characterized by: (1) disorientation, stupor, coma; (2) hypotension; (3) bradycardia; (4) hypothermia.

Surgical shock was ruled out in all cases by hematocrit, red blood cell count and hemoglobin determinations. In 13 of the 46 patients with cervical injuries the syndrome was complete; in 13 not all features were present. In the remaining 20 cases (43.4 per cent) this syndrome was not seen (Table 1).

No definite correlation could be established between the cervical segment traumatized, the nature of the injury, i.e. transection, contusion or concussion, and the occurrence of the syndrome. In the 46 cases observed the lesions were placed as follows:

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Cervical Segment	*Type of Injury*
C1	Contusion 1
C2	None
C3	Transection 1 (died), contusion 1
C4	Contusion 3
C5	Contusion 13, transection 1
C6	Contusion 10, transection 4
C7	Contusion 9, concussion 1, transection 2

It is, of course, impossible to determine the rostral or caudal extension level of injury in these cases. Two of the patients observed in this series

TABLE 1

Case	Level	Injury	Disorientation	Hypotension	Bradycardia	Hypothermia
1	C4	Contusion	Extreme	Extreme	Extreme	Extreme
2	C6	Transection	Extreme	Extreme	Extreme	Extreme
3	C6	Transection	Extreme	Extreme	Extreme	Extreme
4	C5	Contusion	Moderate	Extreme	Extreme	Moderate
5	C5	Transection	Moderate	Moderate	Extreme	Moderate
6	C4	Contusion	Moderate	Moderate	Moderate	Moderate
7	C5	Contusion	Extreme	Moderate	Moderate	Moderate
8	C7	Transection	Extreme	Extreme	Moderate	Moderate
9	C3	Transection	Extreme	Extreme	Extreme	Extreme
10	C6	Contusion	Extreme	Extreme	Extreme	Extreme
11	C5	Contusion	Moderate	Moderate	Moderate	Moderate
12	C7	Contusion	Moderate	Extreme	Moderate	Mild
13	C5	Contusion	Extreme	Extreme	Extreme	Extreme
14	C4	Contusion	Intermittent	Absent	Absent	Absent
15	C5	Contusion	Extreme	Absent	Absent	Absent
16	C4	Contusion	Moderate	Absent	Absent	Absent
17	C6	Contusion	Mild	Mild	Mild	Absent
18	C7	Concussion	Moderate	Absent	Absent	Absent
19	C5	Contusion	Moderate	Extreme (Renal Shutdown)	Mild	Mild
20	C5	Contusion	Extreme	Moderate	Mild	Extreme
21	C5	Contusion	Convulsions	Absent	Absent	Absent
22	C7	Transection	Moderate	Absent	Absent	Absent
23	C1	Contusion	Extreme	Absent	Absent	Absent
24	C7	Contusion	Extreme	Absent	Absent	Absent
25	C6	Contusion	Moderate	Moderate	Absent	Absent
26	C5	Contusion	Nocturnal	Absent	Absent	Absent

Case 9, and Case 20 whose injury was complicated by severe third-degree burns, succumbed.
All patients demonstrated absence of shivering and sweating below the level of the lesion.
All patients showed a moderate amount of abdominal distention.

succumbed. Injury in one was complicated by severe third-degree burns; the second death occurred with transection at the C3 level. This, however, is no index of the severity of the disability in this type of injury, nor does it represent a true index of the mortality or survival rate in cases of severe penetrating wounds of the neck since only the cases in which the patients survived evacuation and reached neurosurgical installations at Mobile Army Surgical Hospitals are recorded. The occurrence of the syndrome is

not an indication of transection, for the bulk of the group (80 per cent) represented contusion and physiological, rather than anatomical, transection of the cord. In all cases the patients were rendered immediately quadriparetic or quadriplegic and regained motor function to a variable degree depending upon the permanent anatomical damage. The components of the syndrome will be considered individually as they may present certain diagnostic and and therapeutic pitfalls.

A. *Disorientation, stupor, and coma.* Psychic changes are marked immediately after injury and may represent interruption of sympathetic components of emotional expression initiated in the hypothalamus.[8,9] The patient may be triaged with a presumptive diagnosis of cerebral concussion, or labelled a psychiatric casualty, as was a soldier with a penetrating gunshot wound of the neck, a comminuted, depressed fracture of C6–C7 and contusion of the 7th cervical cord segment. The patient was evacuated quadriparetic through the first echelon of neurosurgical care, but with disorientation and facetious euphoria as the most prominent features of his clinical picture (Case 24). It was not until he had been transported without cervical support and labelled "conversion hysteria" that lumbar puncture and review of the x-rays demonstrated partial subarachnoid block. The acute anxiety that one might expect in quadriplegia with sudden threat of complete immobility is frequently absent and replaced early by passive, jocular, bland indifference. Stupor, coma and generalized convulsions occurred in a few patients with contusion of the cervical cord.

B. *Bradycardia.* The slowing of the pulse is probably caused by interruption of the preganglionic fibers of the sympathetic cardiac accelerator nerves. The resulting sympathetic blockade may lead to unchecked parasympathetic activity. A 2:1 ratio between heart rate and peripheral pulse rate during the early phase of the syndrome has been observed in a few cases. The bradycardia may be misinterpreted as a reflection of increased intracranial pressure and one of the patients in this series was subjected to diagnostic burr holes because of this misinterpretation (Case 10).

C. *Hypotension.* The loss of peripheral vascular resistance secondary to the absence of sympathetic vascular tone is responsible for the hypotension. The initial impression of "shock" without concomitant blood loss may lead to ill-advised rapid transfusions which succeed only in further loading an inert peripheral vascular pool and as a rule do not raise the blood pressure. Electrocardiographic tracings obtained from these patients 7 to 10 days after injury show normal conduction patterns with sinus bradycardia.

D. *Hypothermia, shivering and sweating.* Sherrington observed in his cervical dogs that shivering and piloerection occurred only in regions innervated above the level of cord section.[7] The weather in Korea has been cold during some of the bitterest fighting and because of the absence of shivering and movement below the level of lesion, patients with cervical injuries have rapidly become chilled and have been admitted with body temperatures as low as 92°. The oral temperature may be a degree or two

higher than the rectal temperature but usually both are markedly depressed. Since sweating too is absent below the lesion, these patients during the summer months may rapidly become hyperthermic.

E. *Gastric distention and ileus.* Temporary neurogenic ileus is a common complicating feature of quadriplegia. We have not seen hyperactivity of the bowel following this type of preganglionic ablation of the sympathetic enteric supply.

TREATMENT

Treatment begins with recognition of the syndrome. These patients tolerate evacuation poorly and care in emergency stabilization of the neck by litter support and improvised cervical brace can be lifesaving. Temperature should be checked frequently and controlled by external heat or, in summer, by cool sponging. Brisk rubbing of the skin in hyperthermic patients may cause sufficient cutaneous dilatation to effectively cool by convection. The level of injury may frequently be plotted on the skin by flush and sweating above the lesion and dry, mottled cyanotic skin below. Care must be exercised in treatment with heat, or in sponging, to keep the skin intact. Burns or abrasions in quadriplegics are notoriously slow to heal and may be the precursors of large decubiti. With quadriplegias, care on a Stryker frame with frequent position change, tidal bladder irrigation and meticulous skin care is mandatory. It is felt that surgical decompression of the spinal cord at the earliest possible instance is indicated in all of these cases.[2] The occurrence of the syndrome does not contraindicate surgery; all of these patients were operated upon under local anesthesia on their Stryker frames. Occasionally the vital signs revert to normal dramatically after decompression of the cervical cord; more often there is a gradual return to normal of the pulse rate and body temperature over a period of days. The reversal to normal in body temperature occurs in some cervically transected animal preparations by acceleration of the basal metabolic rate.[3] This has not been true of those in this series who have had BMR determination after injury (2 cases). No effort has been made to test response of these patients to temperature change after injury since it was not considered in the best interest of the patients to do so in the early convalescent stage. Clark has shown that cats with transection of the lower cervical cord are unable to make the adjustments necessary for maintaining a normal body temperature when there is a sudden and considerable fall in the environmental temperature.

The syndrome appears clinically to be a reflection of a blockade of sympathetic efferent pathways in the cervical spinal cord. Since unbalanced parasympathetic function may accentuate the signs and symptoms, atropine in small doses (0.4 mg.) has been used intravenously in the treatment of some of these patients. No effect on temperature or blood pressure resulted. Striking improvement in the sensorium was reported after its use in Case 2, who had a complete transection at C7. Atropine frequently in-

creased the peripheral pulse rate. In patients with a 2:1 ratio between heart and radial pulse rate, atropine renewed the normal ratio, but relative bradycardia persisted.

CASE REPORTS

Case 4. Tokyo Army Hospital.

A 23-year-old infantry corporal was injured in a truck accident in South Korea on Feb. 14, 1952. He was immediately rendered quadriplegic. X-rays of the cervical spine taken at a mobile field hospital revealed a dislocation of C6 on C7. The patient was transferred the same day to a U. S. Army neurosurgical unit at a mobile surgical hospital. Examination revealed quadriplegia with a sensory level at C5, disorientation, temperature of 92°, pulse 34 and B.P. 80/40. Respirations were shallow and there was marked gastric distention. Queckenstedt was normal but CSF was xanthochromic. Patient was treated supportively and x-ray examination of the cervical spine was repeated. The dislocation could not be visualized and the patient was thought to have had a temporary dislocation that had reduced itself. After being kept under surveillance for several days he seemed somewhat improved and was evacuated by air to Japan. In the short trip by ambulance to the airstrip on a litter without neck support, he suddenly became worse. The blood pressure fell, disorientation was more marked and respirations became shallow with resulting cyanosis. His evacuation was interrupted for several hours because of apparent complete circulatory collapse. He arrived at Tokyo Army Hospital on February 18.

Examination. The patient was semimoribund. Oral temperature was 94°, axillary 92° and rectal 92°. Pulse 46, respirations 20 and B.P. 78/52. There was marked gastric distention. The skin of the face and neck was flushed and moist while below the clavicle it appeared mottled, pallid and dry. The pupils were miotic and responded to light. There was complete motor and sensory loss below C5. The intake for the previous 24 hours had been 3000 cc. and the output 100 cc. The NPN was 58 and the circulating eosinophil count 5.5. The urine had a specific gravity of 1.017 and contained a trace of albumin. EKG showed sinus bradycardia without conduction deficit. X-rays of the cervical spine revealed a dislocation of C6 on C7.

Operation. After 24 hours of Wangensteen suction to a Levine gastric tube for bowel decompression, a cervical laminectomy of C5, C6 and C7 was done under local anesthesia on the Stryker frame. The laminae of C5, C6 and C7 were found to be comminuted and the cord was angulated at the C6 level because of posterior displacement of C7. The cord was contused and appeared softened. Crutchfield tongs with 12 pounds of traction were applied at the conclusion of the operation. During the procedure the blood pressure became imperceptible, but responded to intravenous neosynephrine. With decompression of the cord blood pressure and pulse rate rose.

Course. Immediately postoperatively B.P. was 130/60 with pulse of 84 and temperature of 97°. The 24-hour urinary output for the first postoperative day was 2800 cc. with an intake of 2500 cc. Thereafter the pulse and blood pressure remained within normal ranges and the body temperature slowly reverted to low normal limits. The BMR 10 days postoperatively was −5. On February 28 slight motion of the arms and right leg was noted. The patient was evacuated to the Zone of the Interior on April 10 with 30 per cent function of the arms and normal vital signs. Prior to evacuation he had been sitting up with a cervical brace.

Case 1. 1st Prov. Neurosurgical Detachment, Korea.

A 24-year-old Marine private sustained a penetrating bullet wound of the neck in action in North Korea on April 24, 1951. There was immediate flaccid quadriplegia and anesthesia below the C4 dermatome. There was no Horner's syndrome. Patient was conscious. All deep tendon reflexes and superficial reflexes were absent. The pulse was 42; B.P. was 80/65. X-rays of the cervical spine revealed a fracture of C4.

Operation. On April 26 a cervical laminectomy of C4 and C5 was done under local anesthesia with the patient prone on a padded litter. A metallic fragment was removed together with the comminuted fragments of the C4 and C5 laminae. The dura was intact and the cord appeared somewhat ischemic.

Course. Postoperatively the patient improved for 24 hours, then became disoriented. Bradycardia, hypotension and hypothermia continued for 72 hours while the patient remained stuporous with periods of disoriented consciousness. Intermittent clonic extensor movements of the neck were noted. Lumbar puncture revealed normal pressure and no subarachnoid block. The patient was treated with external heat, ephedrine, atropine, cervical immobilization, two hourly turning, and constant and complete nasopharyngeal toilet.

The vital signs gradually improved until, by the end of the first postoperative week, they had reverted to normal. The patient was subsequently evacuated to Japan where he was nursed on a Stryker frame and rehabilitation was started. (This is the only case in the series in which the syndrome was observed to occur postoperatively.)

SUMMARY

An acute cervical syndrome, characterized by disorientation, stupor, coma, hypotension, bradycardia and hypothermia, is described in cases of penetrating and closed war injuries of the cervical spinal cord. A discussion of the neurophysiological basis of the findings, and the recognition and treatment of the syndrome is presented. Early cautionary and supportive measures and prompt surgical decompression of the spinal cord are recommended.

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FURTHER OBSERVATIONS ON MEDULLARY SPINOTHALAMIC TRACTOTOMY*

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SINCE our previous report[1] there has been considerable interest in the problem of medullary spinothalamic tractotomy for the relief of pain. We feel it would be valuable to report additional cases and to give a more detailed description of the technique employed. Special reference will be made to 3 of the cases, because in them there occurred some trigeminal sensory changes which are of interest anatomically.

In our previous communication, in February 1947, we reported 11 cases in which the operation was performed for relief of arm and shoulder pain. Three of the patients died early postoperatively. All patients obtained satisfactory analgesia up to the C3 or C2 levels. D'Errico[2] and Klemme[5] have described similar satisfactory experiences.

We are reporting 8 additional cases of unilateral, intractable shoulder-arm pain. In 6 of these cases the pain was caused by metastatic carcinoma. In the other 2 the pain was from non-malignant causes. Three of the 8 patients died before discharge from the hospital. All had extensive general and pulmonary malignancy. One died suddenly and unexpectedly from pulmonary embolism on the 10th postoperative day. The others lived either to die of their malignancy or are still alive and largely relieved of pain.

TECHNIQUE

During surgery, the sitting position was used for all patients except one because it seemed to one of us (A.S.C.) that the ease of operating thus outweighed the risks entailed, viz. air embolism, operative shock and subsequent hemorrhage. However, operative shock and subsequent headache from aspirated subarachnoid air are less apt to occur in the horizontal position, and this is the method of choice in those cases in which the risks are more serious.

We prefer to operate under local infiltration and block anesthesia together with appropriate sedation. A few patients were so apprehensive that intravenous sodium pentothal was given. The patients were permitted to regain consciousness at the time of the medullary section. Their reactions were not as accurate as they would have been had the operation been done completely under local anesthesia, but the results were satisfactory.

The incision (Fig. 1) has been inverted U-shaped, extending from below and just medial to the tip of the mastoid process and arching upward and

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medially and then downward in the midline to about the level of the 4th cervical spine. The highest part of the incision is about the level of the line of insertion of the suboccipital muscles. These muscles should be cleanly dissected away from the occiput, exposing the atlas.

The ring of the atlas is resected from midline laterally to the edge of the groove of the vertebral artery. This artery should be visualized and protected with a thin spatula to prevent injuring it with the rongeur.

The foramen magnum should be enlarged by removing sufficient bone so that the cerebellar tonsil can be elevated to visualize the obex, which is an essential landmark. After the dura has been opened, the arachnoid is punctured and cerebrospinal fluid drained out and the area kept dry with suction.

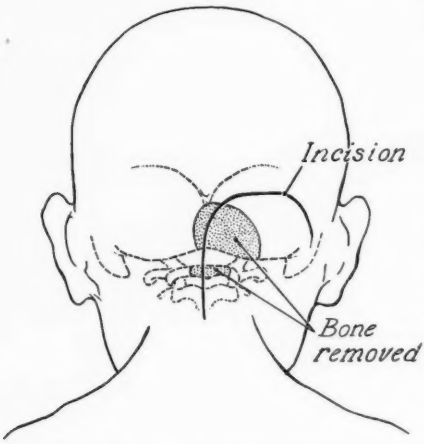

FIG. 1. Diagram showing location of skin incision and amount of bone removed.

To visualize the lateral wall of the medulla, the posterior inferior cerebellar and vertebral arteries may need to be pushed laterally. They should be protected with a thin cottonoid strip. Often there are a number of fine attachments of arachnoid which have to be carefully cut with fine scissors. If the patient is conscious and feels pain at this stage, the area can be anesthetized, using small flat oblong cottonoid stamps moistened, but not dripping, with 1 per cent novocain solution.

One must be certain that the site of puncture is caudad to the lower pole of the olive. The olivary eminence can usually be seen by looking ventrally beyond the line of emergence of the rootlets forming the 10th and 11th cranial nerves. If it is not safe to force a view anteriorly to visualize this, one can fairly safely be guided by the obex and the nerve rootlets. The site of puncture is in the lower medulla cephalad to the decussation of the pyramids. This site is usually 3 to 4 mm. caudad to the obex near the junction of the rootlets forming the 10th and 11th cranial nerves and just ventral to them (Fig. 2). These rootlets emerge from the medulla immediately ventral to the eminence of the spinal tract of the trigeminal nerve. The dentate ligament cannot be used as an accurate guide at this level because it has already terminated.

The instrument for the medullary incision is optional. We use a set of four knives made for this special operation. The cutting blade is about 1 mm. wide and is set at a 90° angle and tilted about 30° upwards in the long axis of the handle. Two are sharpened on the underside with a slight hook

FIG. 2. Site of puncture 3 or 4 mm. caudad to the obex and ventral to the tuberculum cinereum.

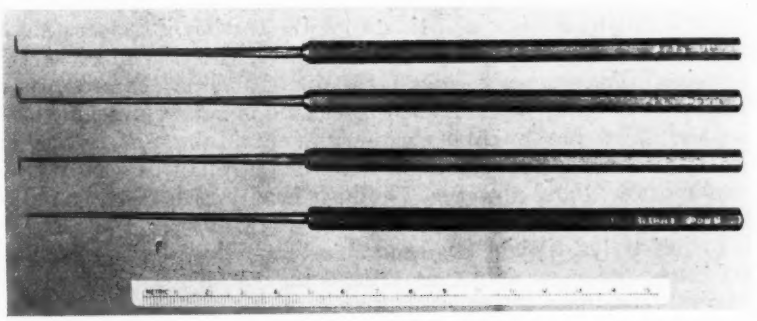

FIG. 3. Upward and downward cutting knives for medullary spinothalamic tractotomy.

downwards and two are similarly made for upward cutting (Fig. 3). Fig. 4 shows the location and area of cutting in one case. We feel that ataxia is largely avoided by cutting caudad to the olive and by minimal destruction of the spinocerebellar pathways made possible by these special knives. Fig. 5

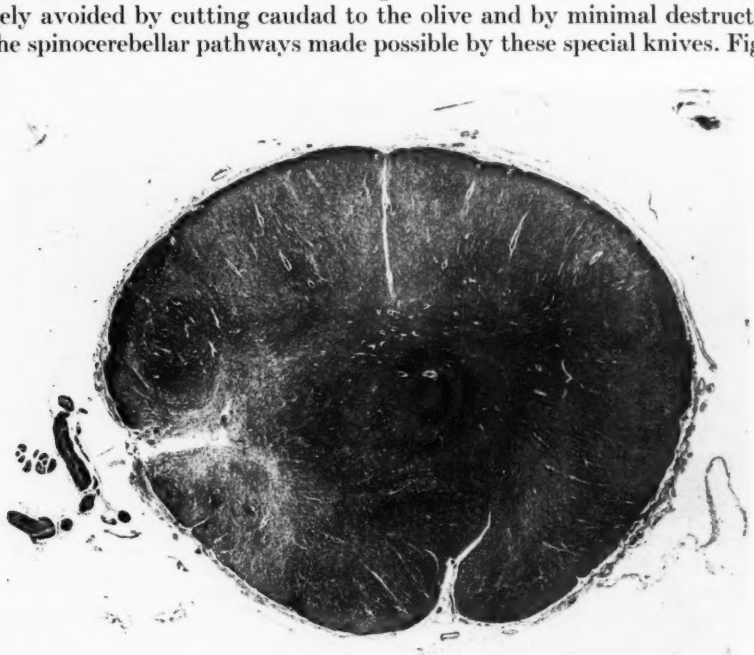

FIG. 4. Photomicrograph of lower medulla showing site of medullary spinothalamic tractotomy and extent of damage to surrounding structures in Case 18. (Fiber stain)

shows the anatomical structures more diagrammatically, illustrating the amount of tissue usually cut by the up-cutting and down-cutting knife blades.

DISCUSSION

There is usually little or no pain during the cutting, if the area has been locally anesthetized, and the descending root of the trigeminal nerve has been avoided. There is no bleeding if the site of puncture has been touched with the Bovie, using the minimal current. It is our impression that the inward and upward cutting was necessary to obtain the high cervical level. We have tried differential cutting in only one case. In this, only one cut was made inwards and upwards. The high level was apparently obtained but was maintained for only 1 month.

D'Errico[3] stated that he has been able to avoid analgesia of the lower part of the body but we do not have the details of his technique. It will be a desirable refinement if analgesia of the trunk and lower extremity can be avoided.

In addition to the high cervical levels obtained from section of the spinothalamic tract, some sensory changes indicating injury to the trigeminal pathways were noted in 3 cases.

These sensory changes are of special interest for, in addition to the ipsilateral analgesia in the area of distribution of the trigeminal nerve, which

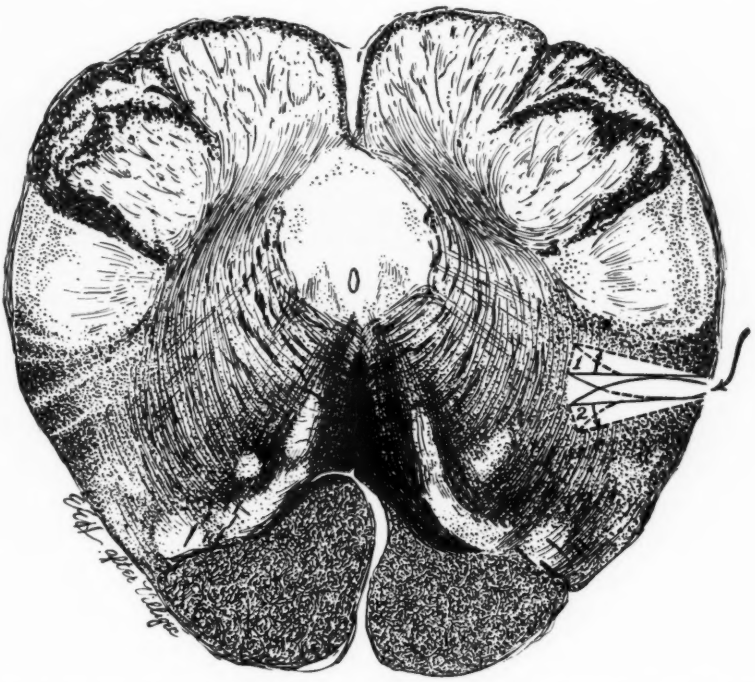

FIG. 5. Diagram showing the method of section of the spinothalamic tract in the medulla by the up-cutting and down-cutting knife blades. The point of entry is approximately 1 mm. in width, thus largely sparing the fibers of the spinocerebellar tracts.

might be expected from damage to the descending root, contralateral analgesia was also obtained. The loss of sensation to pain was complete on the ipsilateral side early, involving all three divisions of the trigeminal nerve. On the contralateral side in 2 cases there was a similar but smaller area of pain loss, occupying primarily the ophthalmic but also the mesial portions of the maxillary and mandibular divisions. In the third case the ophthalmic division was primarily involved.

On both sides of the face as pain sensation returned, it did so in an orderly fashion; first to the mandibular region, then maxillary, and finally, but not completely, to the ophthalmic division. In addition to this orderly return from lower to upper face there was also suggestion of segmental

innervation in that the sensation to pain returned more rapidly in the perioral and perinasal regions than it did peripherally. A somewhat similar observation was noted by Hyndman[4] in his study of differential section of the posterior root of the trigeminal nerve.

The loss of pain sensation on the contralateral face was less than on the ipsilateral side and, on both sides, was most marked and persisted longer in the ophthalmic division. In 2 of these patients, sensation to pain never returned to the ophthalmic division on either side, although the corneal reflex was preserved bilaterally.

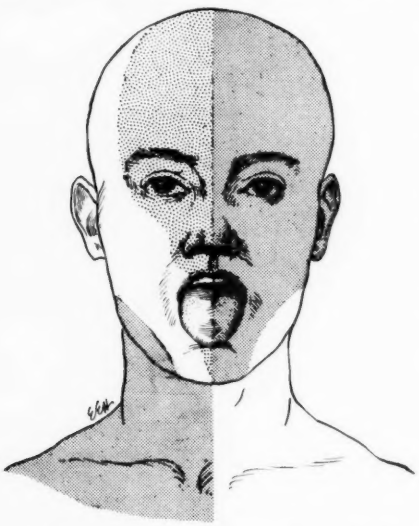

FIG. 6. Diagram showing maximum distribution of loss of pain and temperature sensation following medullary spinothalamic tractotomy in Case 18.

One of these patients (Case 18), whose sensory changes are illustrated in Fig. 6, died 3 months following surgery and Fig. 4 shows the area of maximum destruction in the medulla. There is complete destruction in the spinothalamic tract and considerable damage to the surrounding area of reticular formation mesially, and to the ventralmost fibers of the descending root of the trigeminal nerve.

From these findings it would seem that the loss of pain sensation for ipsilateral face is caused by destruction or injury of the fibers of the descending tract of the trigeminal nerve and to its nucleus, and that the arrangement of these fibers from a dorsal to ventral position in the descending root is mandibular, maxillary and ophthalmic, with the ophthalmic fibers lying just above the spinothalamic tract.

Contralateral sensory disturbance for face is assumed to be caused by involvement of the central trigeminal tract after it has crossed from the spinal nucleus of the trigeminal nerve opposite the site of section in the medulla, and is ascending upwards to the thalamus. This would suggest that the central trigeminal tract courses upward in a position just medial to the cervical fibers of the spinothalamic tract, with the fibers of the caudal or ophthalmic portion of the nucleus lying nearest the spinothalamic tract and the maxillary and mandibular fibers medial to these (Fig. 7). Stopford[6] noted similar sensory changes in supposed cases of posterior inferior cerebellar artery occlusions and placed the central trigeminal tract in a similar position medial to the spinothalamic tract. Since the medullary section was made just cephalad to the cervical-medullary junction, it would

seem that some of the maxillary and mandibular fibers as well as the ophthalmic fibers of the descending root of the trigeminal nerve descend to and enter the caudal-most portion of the spinal nucleus of the trigeminal nerve.

A brief summary of the 8 cases follows.

CASE REPORTS

Case 12. F.L., a young woman of 28 with intractable pain from a severe obliterating endarteritis of the body, but worse in the left arm, had not responded to medical measures. Sympathectomy did not help. Right medullary spinothalamic tractotomy

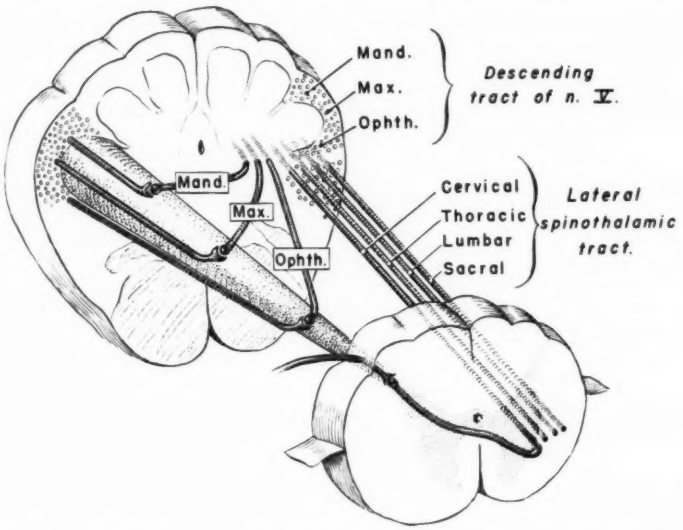

FIG. 7. Diagram illustrating anatomical relationships of descending tract of trigeminal nerve, medullary spinothalamic tract and central trigeminal pathways.

was done Mar. 12, 1948 with relief from most of the pain. It did not solve all of her problems but did make life bearable.

Case 13. A.Y., a man of 60, was suffering from severe traumatic causalgia of the right upper extremity. Pain was not relieved by dorsal sympathectomy. The patient was completely disabled for over 2 years. A left medullary tractotomy was done Sept. 24, 1948. Relief of the worst pain resulted and a level of complete analgesia at C2 segment has persisted. He has been able to work part time, but still complains of a burning sensation all over his body and is still a problem to his employer.

Case 14. A.G., a woman of 35, suffered from intractable pain of the right axilla and arm from inoperable carcinoma. Left medullary spinothalamic tractotomy was done Jan. 18, 1949. She died 10 days later from thrombosis and abscess formation associated with extensive malignant involvement of the lungs. She had been relieved of pain and had had a high level of analgesia.

Case 15. A.M., a man of 50, had intractable pain of the left axilla, shoulder and arm caused by inoperable carcinoma. A right medullary tractotomy was done Oct. 28, 1949. He was relieved of his pain and had analgesia up to C3 level. He returned to the hospital just before his death Jan. 24, 1950, the result of the carcinoma.

Case 16. R.H., a woman of 59, was suffering from intractable pain of the right arm and shoulder from inoperable carcinoma. A left medullary tractotomy was done Aug. 2, 1950 with relief of the pain and giving a level of analgesia at C3. She died suddenly on the day of her proposed discharge, 10 days postoperatively, from acute pulmonary complications of her lung carcinoma.

Case 17. M.I., a colored man of 70, had intractable pain of the left arm and shoulder from inoperable carcinoma. A right medullary tractotomy was done Aug. 11, 1950 with relief of pain and a C3 level of analgesia. He, however, did not recover fully from his operation and died Aug. 15, 1950. He showed extensive involvement of both lungs, and other organs. It is possible that he might have survived longer had we avoided severe shock by operating upon him in the horizontal lateral position.

Case 18. J.K., a man of 45, had intractable pain of the left arm from a Pancoast tumor of the left apex with involvement of D2 vertebral body. A right medullary tractotomy was done Nov. 3, 1950 with satisfactory relief of his pain on the left side. A left medullary tractotomy was discussed but decided against. His pain was ameliorated by hypnotics and Proteinimine but he rapidly became weaker and died 3 months after the tractotomy. At autopsy, the spinal cord and brain were removed. Fig. 4 shows a section of the lower medulla at the site of cutting.

Case 19. G.C., a woman of 60, had intractable pain from inoperable metastatic carcinoma of the left axilla and shoulder. A right medullary tractotomy was done July 25, 1951. The patient was in the recumbent position; her condition was excellent throughout the procedure. She cooperated unusually well under local infiltration and block anesthesia.

The up-cutting knife was entered and one cut was made medially and dorsally. Following this she seemed to have complete analgesia up to the C2 level, including the lower part of the body.

About 1 month after the operation, she began to experience pain in the axilla and shoulder and tests with a pin showed that the analgesia below D10 level was still absolute but that it was only partial above D7 level. It was apparent that the entire tract had not been severed. We have offered to make a deeper cut if the pain becomes unbearable but she has not yet presented herself for this to be done.

SUMMARY

We have reported 8 additional cases of intractable pain in upper levels treated by medullary spinothalamic tractotomy. We feel that this is the preferable surgical procedure for treating intractable shoulder-arm pain. We believe that the experiences of other neurological surgeons will corroborate our results and that this operation should become the generally accepted procedure for this condition.

The associated trigeminal sensory changes were pointed out to help settle the exact anatomical relationships in this area.

It would be even more desirable if a subtotal differential section of the spinothalamic or of the trigeminal tracts could be made in the medulla which would give long-lasting results.

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THE SYNDROME OF TRAUMATIC INTRACEREBELLAR HEMATOMA

WITH CONTRECOUP SUPRATENTORIAL COMPLICATIONS

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In spite of the large numbers of head injuries that are observed in the hospital wards and in the autopsy rooms, relatively little neurosurgical data are available on traumatic lesions of the posterior fossa. During the past two years 8 lesions of this type have been operated upon at the University Hospital. In this group of head injuries were 4 patients with traumatic intracerebellar hematoma with associated contrecoup supratentorial complications, either intracerebral hematoma or severe contusion. Certain concepts evolved which have proven of value in formulating a new policy in the treatment of these head injuries, and many of them previously considered inoperable may in fact be treated surgically with a good prognosis.

Our interest was aroused in traumatic lesions of the posterior fossa when we encountered our first case of extradural hematoma of the posterior fossa.[23] A woman was admitted to University Hospital with a history of having struck her head in a fall in the bath tub 5 days earlier and of being unconscious for 4 hours after the injury. She sustained a midoccipital stellate laceration of the scalp, and then signs and symptoms of a left posterior fossa lesion developed. There was no roentgenographic evidence of skull fracture. At operation a large extradural hematoma was removed from the left posterior fossa and the patient recovered.

After examining the case histories of extradural hematoma of the posterior fossa that were available in the literature[1,2,4,6,8–10,13,15,21,24–26] it became apparent that many individuals with this type of lesion had no signs or symptoms referable to the posterior fossa and suddenly died a respiratory death without significant change in vital signs to warn of the impending disaster. The diagnosis of an expanding traumatic lesion of the posterior fossa depends upon the suspicion that such a lesion exists. This has been emphasized by several authors. If the symptoms of a posterior fossa lesion such as ataxia, nystagmus, dysmetria, etc. are present, attention is directed to that region. However, we suggested in our first report[23] that since a definite syndrome cannot be relied upon for establishing the diagnosis, objective findings must be depended upon. A laceration or contusion over the occiput plus a lambdoidal suture line separation or fracture line traver-

* Now in military service, U. S. Army.

ing the lateral sinus or extending through the foramen magnum are suggestive findings. In a patient who has had an increasing depth of unconsciousness and no other evidence of a supratentorial lesion these are sufficient to warrant trephination over the posterior fossa.

Using these criteria for operation we discovered 3 more patients with surgically significant extradural hematomas of the posterior fossa, one confined to that region and the other two extending upward across the transverse sinus to include the occipital area. All 3 of these patients made rapid, uneventful recoveries; their course has been described elsewhere.[16]

The use of our criteria also resulted in finding intracerebellar hematomas in 4 additional patients with associated contrecoup intracerebral clots or severe contusions. These cases we wish to report in detail.

CASE REPORTS

Case 1. V.H., 39 years old, a white male alcoholic, had fallen and fractured his left wrist 10 days prior to admission. It was not known whether he had injured his head, but he was ill in his rooming house for a few days. He recovered sufficiently to walk on the street where he staggered, fell again striking his head, became stuporous, and was placed in the local jail for drunkenness. Tremors of all four extremities developed, and he was semicomatose for 4 or 5 days. A lumbar puncture yielded a grossly bloody CSF with a pressure of 250 mm. of water. The patient was transferred to University Hospital.

Examination. His temperature was 104°, pulse rate was 140/min., respirations were 36/min., and B.P. was 198/100. The patient was acutely ill, incontinent of urine, and semicomatose, but could be roused sufficiently to state his name and occupation. His pupils were small, equal and did not react to light. Extraocular movements were normal. No nystagmus was observed. There were hypoactive deep tendon reflexes on the right side, positive extensor plantar reflexes bilaterally and episodes of generalized fine tremors. Roentgenograms of the skull revealed a linear fracture in the left suboccipital area (Fig. 1). A diagnosis of possible posterior fossa extradural hematoma was proposed.

1st Operation. Burr holes were made bilaterally over the posterior fossa and above the transverse sinus. The burr hole at the fracture site in the left suboccipital region revealed a 3–4 cm. laceration of the dura mater near the midline. The left cerebellar hemisphere was exposed, and an area of necrotic contused brain and extruding intracerebellar hematoma, 3 cm. in diameter, was removed. After all bleeding had been controlled the wound was closed without drainage. No abnormality was found either extra- or intradurally at the supratentorial burr holes.

Course. For the first 3 postoperative days the patient reacted well and appeared to be improving, but on June 13th he became irrational, restless and progressively more comatose.

2nd Operation. Trephination was performed in the frontal and temporal areas bilaterally and in the right occipital region. Upon incising the dura at all of these points black liquid subdural fluid and small clots gushed forth. The cortex was severely contused and edematous, and displayed increased intracranial pressure. The most marked changes were in the right temporal region. After the small clots were evacuated the brain did not pulsate nor did it tend to extrude through the burr holes.

Course. The patient improved transiently but 3 days later his condition again declined, the respiratory rate increasing to 30 or 40/min., and on June 19th his respirations ceased.

Postmortem Examination. Both cerebral hemispheres were markedly congested with flattening and broadening of the convolutions. There was a minimal amount of diffuse subdural hemorrhage bilaterally over the hemispheres. There was softening in the frontal lobes with extensive contusion and laceration of the orbital surfaces. On attempting to remove the brain a large clot was expressed from the anterior pole of the right frontal lobe. Minor intracerebral hemorrhages were also found in the tips of the left temporal and frontal lobes (Fig. 2). There was marked edema of the medulla.

FIG. 1. *Case 1.* Roentgenogram of skull demonstrating linear fracture in left occipital bone.

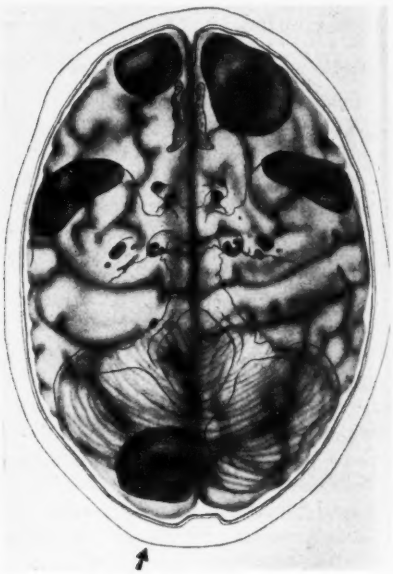

FIG. 2. *Case 1.* Diagram indicating site of the left intracerebellar hematoma, the large contrecoup right frontal intracerebral clot, and minor clots in the temporal and left frontal lobes.

Comment. Although there had been severe destruction of the left cerebellar hemisphere there was little tendency for it to herniate through the dural tear, and it was believed that the lesion might not have been of surgical significance. Nevertheless after removal of the destroyed brain and intracerebellar clot there was considerable improvement in the patient's condition. This could have been on the basis of internal decompression permitting expansion of the intracerebral hematomas in the temporal and frontal regions, particularly the one in the right frontal area. The case emphasizes the importance of searching for a contrecoup lesion. In retrospect we probably should have explored the right frontal and temporal areas more

thoroughly. At the time of the second operation bilateral subtemporal decompressions might have been made, but it is doubtful that the patient could have survived considering the extensive cerebral damage noted at autopsy.

Case 2. H.B., 53 years old, right-handed white female, was involved in an automobile accident at 4:30 P.M. on Sept. 18, 1951, when another car struck the one in

FIG. 3. *Case 2.* Roentgenogram showing midoccipital linear fracture traversing the right lateral sinus and extending into the right suboccipital area (retouched).

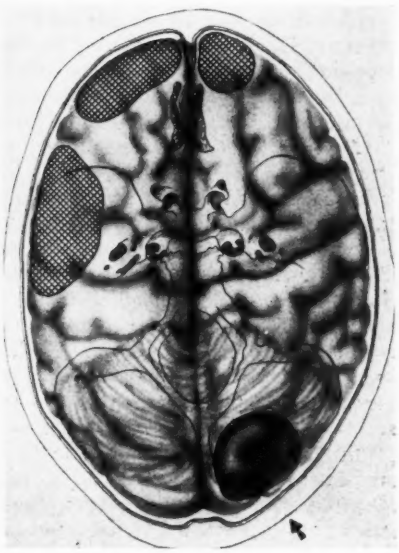

FIG. 4. *Case 2.* Diagrammatic representation of right intracerebellar hematoma and the major contrecoup temporal and frontal contusions with minor contusion of the right frontal area.

which she was riding. The door was forced open and she fell out, striking the back of her head against the curb. The patient was not knocked unconscious but severe generalized headache and projectile vomiting rapidly developed.

Examination one hour later at University Hospital revealed the B.P. to be 120/80, pulse rate 72/min., and respirations 26/min. There was a 4.5 cm. laceration of the scalp in the midoccipital region. The patient was lethargic and disoriented with regard to time and place. The pupils were equal and extraocular movements were normal. Nystagmus was observed on right lateral gaze. There was ataxia on right finger-to-nose test. Roentgenograms of the skull demonstrated an extensive linear occipital fracture close to the midline but extending slightly to the right across the transverse sinus into the right posterior fossa (Fig. 3). Because of the patient's progressive lethargy, severe headache and vomiting, neurologic findings, and roentgenograms a tentative diagnosis of right posterior fossa extradural hematoma was made.

Operation. At 9:00 P.M. that evening the occipital laceration was debrided and

the underlying linear fracture line was exposed. A burr hole next to the fracture line in the right suboccipital region was enlarged to include removal of the rim of the foramen magnum. A 3 cm. laceration of the dura mater was found with a moderate amount of cerebellum extruding through it under marked pressure. The dural opening was enlarged and there was prompt herniation of necrotic cerebellum and intracerebellar hematoma. Several small cortical vessels were seeping blood and one bridging vein near the transverse sinus was actively bleeding. About one-third of the right cerebellar hemisphere had to be sacrificed (Fig. 4). When the superior portion was excised there was a sudden release of cerebrospinal fluid under increased pressure. The basal cisterns were opened and the dural incision was not closed. Burr holes in the left suboccipital area and in both occipital regions above the sinus disclosed no extra- or intradural abnormality. The brain pulsated freely and did not herniate. All wounds were closed without drainage.

Postoperative Course. The patient was irrational, tearing off her head dressing, removing her Foley catheter, and 36 hours after operation roamed the hall so that it was necessary to sedate and restrain her. A week later she was much clearer mentally and began to take fluids orally. A lumbar puncture on October 4th yielded a clear fluid under 150 mm. water pressure.

On the day of discharge 20 days after injury an EEG was made (Fig. 5) which showed a marked left temporofrontal abnormal pattern. At this time there was mild aphasia with slight mental confusion and a tendency toward repetition of phrases, but usually her conversation was intelligible. She had a suggestive right 6th cranial nerve palsy, walked poorly with a wide base, but the dysynergia and dysmetria in her upper right extremity had disappeared. The deep reflexes were equal and active, and there were no pathological reflexes.

On follow-up examination a month later it was discovered that she had no memory of her hospital stay. She had occasional difficulty in expressing herself, and had noted anosmia since her accident. The right pupil was slightly larger than the left, but both pupils reacted to light and in accommodation. Nystagmus was observed on left lateral gaze. Reflexes were slightly hyperactive but equal.

Subsequent examination revealed some euphoria but otherwise neurological findings were negative.

Comment. This patient's right intracerebellar hematoma was expanding rapidly, presenting a true surgical emergency, and it is felt that she would have succumbed from acute medullary compression if she had not been operated upon at once. The postoperative EEG abnormality in the left temporofrontal region (Fig. 5) suggested a contrecoup lesion at that site, possibly concussion, contusion, or a self-limited intracerebral hematoma. The abnormalities in electrical potential might also have been referred to that area because of the damage to the right cerebellum (Bagchi, *et al.*[3]). However, the fact that the patient noted anosmia, loss of memory, and a certain degree of motor aphasia suggested severe local contusion of the left frontotemporal junction as well as the orbital surfaces of the frontal lobes. The important point is the progressive improvement in her EEG and neurologic status.

Case 3. V.S., 34 years old, right-handed white male, was admitted to University Hospital at 9:00 P.M. on Oct. 19, 1951, in a semicomatose condition. At 1:30 A.M.

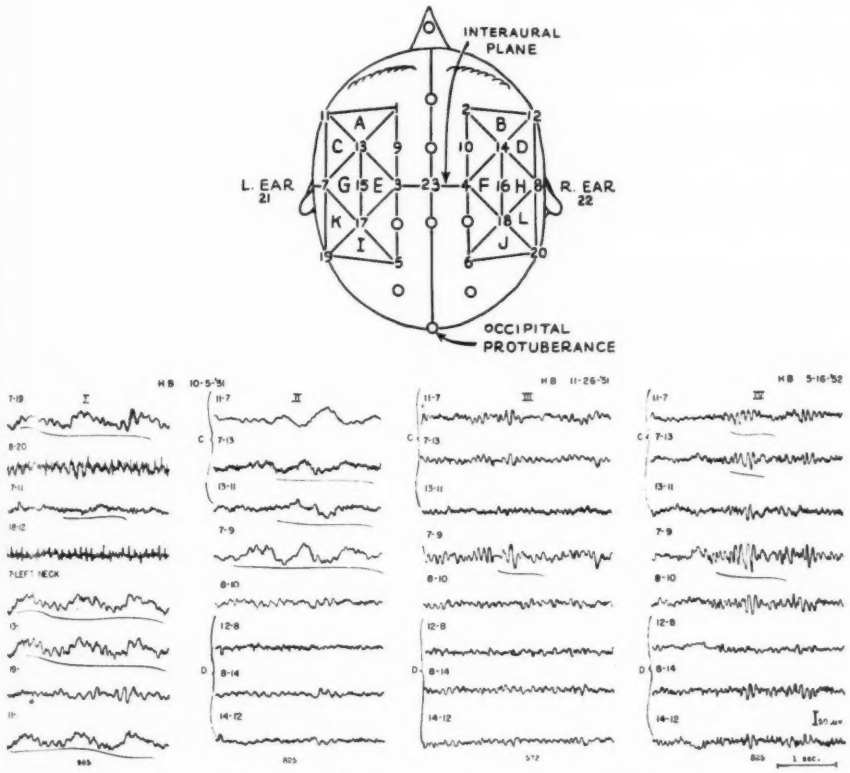

FIG. 5. *Case 2.* Serial EEGs following removal of right intracerebellar hematoma.

Explanation of Fig. 5

(I) 20 days postoperative. Patient is confused but does respond. *Strip 1:* Frequent abnormal slow waves of 1 to 1.5/sec. from left midtemporal region, lead 7, indicating some sort of focal pathology. *Strip 3:* Relative cancellation of slow waves in the linkage of close and equipotential active regions, leads 7 and 11. *Strips 2 and 4:* Right side noncontributory. Fast waves are muscle artefacts. *Strips 5, 6, 7, and 8:* With left neck reference lead same type of abnormal delta waves are found from left midtemporal (7), Sylvian notch (13), anterior temporal (11), but not from posterior temporal (19) lead. Bipremotor bursts (not shown here) may have some deep level significance (Bagchi, *et al.*[3]) in relation to the recent posterior fossa surgery. Significant signs are underlined. Microvolt and time calibrations are given on EEG records.

(II) Same date. Left anterior lateral triangle (C) and left midtemporal premotor linkage (*strips 1 through 4*) frequently show abnormal 1 to 1.5/sec. deltas but not the corresponding triangle (D) and midtemporal-premotor linkage of the right side (*strips 5 through 8*). Left anterior quadrant was more involved than left posterior quadrant (not shown).

(III) 52 days postoperative. Same lead combinations as in II show total disappearance of deltas, indicating marked improvement of the focal condition. Some spiky alpha bursts are still present at lead 7 (*strips 1 and 4*) and some rare undulations of low voltage in 7-3 lead linkage (not shown).

(IV) About 7 months postoperative. Same type of alpha bursts at lead 7, but no focal delta waves. Premotor bursts are absent in 2nd and 3rd postoperative EEGs.

the day before he had been thrown from his car, striking his head on the pavement. He was found in a semicomatose state, and taken to a local hospital where roentgenograms of the skull demonstrated a fracture in the right occipital bone crossing the transverse sinus into the suboccipital area (Fig. 6). The calcified pineal gland was in the midline. His condition declined and he was transferred to University Hospital with a diagnosis of acute subdural hematoma.

Examination. His B.P. was 136/104, pulse rate was 140/min., and respirations were 26/min. The patient was comatose but responded to painful stimuli with purposeful movements of all four extremities. There were multiple contusions and abrasions of the scalp with a particularly large one over the occiput. The fundi were normal. The pupils were small, the right one being slightly larger than the left, but

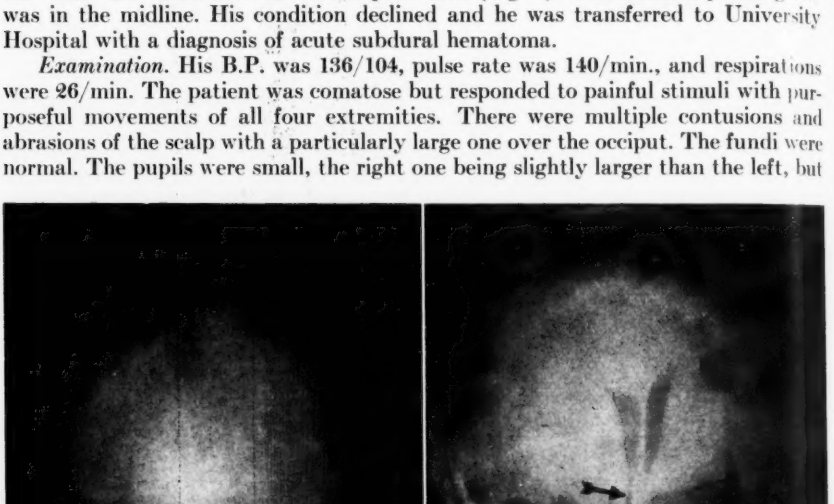

FIG. 6 (*left*). *Case 3.* Roentgenogram exhibiting right linear suboccipital fracture.

FIG. 7 (*right*). *Case 3.* Ventriculogram demonstrating shift of ventricular system caused by left temporal intracerebral hematoma.

both reacted to light. Extraocular movements were intact and no nystagmus was observed. There was an equivocal right extensor plantar reflex.

1st Operation. On the following day the laceration in the occipital region was debrided, and a burr hole was made in the right posterior fossa next to the fracture line. An underlying dural tear was disclosed which was further unroofed by a right suboccipital craniotomy extending through the rim of the foramen magnum. Herniating through the 4 cm. tear in the dura were pulped cerebellum and intracerebellar hematoma. About a third of the damaged right cerebellar hemisphere had to be excised. At the end of the prodecure the brain pulsated well, and cerebrospinal fluid leaked out of the cistern. No attempt was made to close the dura mater and the wound was closed tightly without drainage. Burr holes were then made over the homologous left suboccipital area, over both occipital regions 3 cm. to either

side of the midline, and over the right posterior parietal area 2.5 cm. above and behind the right ear. The dura mater was incised at all regions and no abnormality was found.

Course. Twenty-four hours after operation the patient was much more responsive, obeying simple commands, and even talking slightly on painful stimulation. There was a mild right central facial nerve palsy. On the 6th postoperative day he became aphasic, less responsive, and had difficulty in swallowing. His vital signs were stable, but definite papilledema had developed. The right pupil was larger than the left, but both reacted to light. A right facial palsy was noted. There was increased tonus in the right lower and both upper extremities with sustained right ankle clonus. A diagnosis of left frontotemporal subdural hematoma was made; a repeat roentgenogram of the skull exhibited no significant displacement of the calcified pineal gland to suggest an expanding supratentorial lesion.

2nd Operation. On October 25th a ventriculogram showed a shift of the ventricular system from the left to the right with a curving of the 3rd ventricle indicating a left temporal lesion (Fig. 7). A left temporal craniotomy was performed. Upon opening the dura mater a subdural clot, 0.5 cm. in thickness, was evacuated and a large intracerebral hematoma, 3–4 cm. in diameter, was removed (Fig. 8). A Penrose drain was left in the cavity for 24 hours.

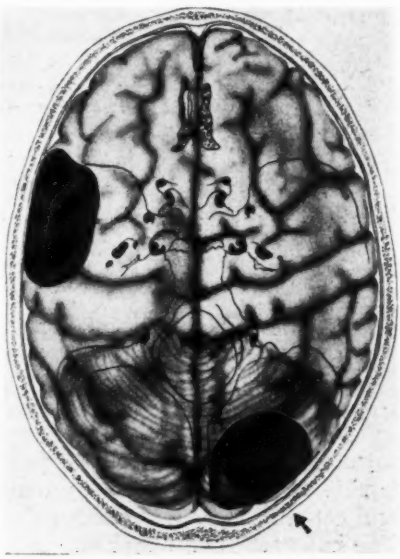

FIG. 8. *Case 3.* Diagrammatic localization of right intracerebellar hematoma and contrecoup temporal intracerebral clots.

Course. On the following day the patient responded to commands, but talked incoherently. Five days later he had Cheyne-Stokes respirations, pulse was 100/min., B.P. was 90/60 and temperature was 98°. After correction of his electrolyte balance by adequate and selective hydration he improved markedly.

At the time of discharge on his 26th postoperative day he could identify objects and use them properly, but was slightly confused as to place. There was no papilledema. The pupils were equal and reacted to light. Extraocular movements were normal. Nystagmus was more pronounced on looking to the left. There was a mild right hemiparesis with a tendency to deviate to the left as he walked. There was slight right dysdiadochocinesis. Muscular tone was normal throughout. The reflexes in the left lower extremity were moderately hyperactive, and an equivocal right extensor plantar reflex was recorded.

At the time of follow-up examination 55 days after his initial operation, an EEG was made, a sample of which is presented (Fig. 9). It showed minor focal changes in the left midtemporal region. Clinically, he exhibited considerable perseveration, impairment of memory, and minimal right hemiparesis, but all posterior fossa signs had disappeared.

Comment. After suboccipital craniotomy with removal of the right

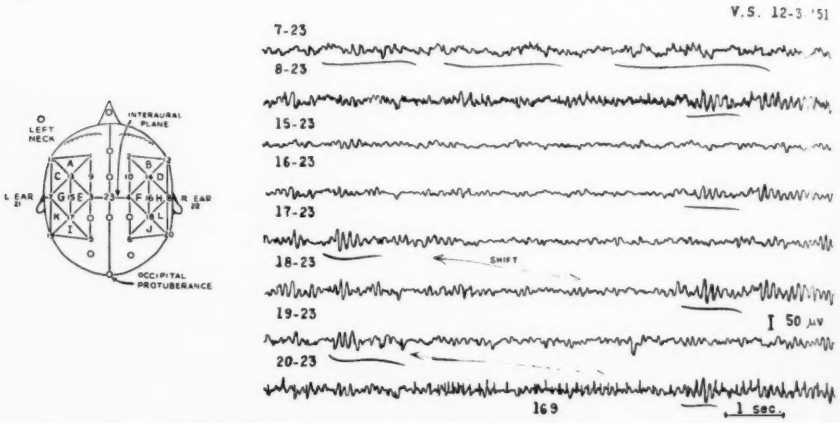

FIG. 9. *Case 3.* EEG 55 days after evacuation of right intracerebellar hematoma and 50 days after removal of left temporofrontal intracerebral clot.

Explanation of Fig. 9

Before the EEG examination patient was ambulatory but slightly confused. The background EEG pattern in most leads is within normal limits. There are, however, rare to occasional single 3 to 4/sec. low voltage abnormal slow waves and undulations in left midtemporal area (*strip 1*, lead 7). These are probably postoperative changes. There is some episodic shift of normal alpha bursts between homologous areas of posterior quadrant of the two hemispheres (underlined portions *strips 2 through 8*). The significance of these cannot be adequately evaluated. This is often seen in posterior fossa conditions (Bagchi, *et al.*[3]).

intracerebellar hematoma the patient improved markedly. The clot was sufficiently large to cause a pronounced degree of medullary compression and could have caused sudden death if it had been permitted to expand. The importance of observing the patient carefully for the development of a contrecoup lesion is well demonstrated. Both intracerebellar and intracerebral hematomas were definite surgical lesions.

Case 4. R.R., 31 years old, a left-handed airplane pilot, was injured at 9:10 A.M. on March 26, 1952. He fell backward from an airport service truck moving at 30 miles per hour and struck the back of his head on the concrete runway. A 4 cm. laceration in the right occipital area was sustained and there was profuse hemorrhage from his nose and ears bilaterally. There may have been a transient period of unconsciousness, but on admission to University Hospital at 10:30 A.M. he was oriented, although suffering from excruciating headache and severe bouts of projectile vomiting.

Examination. His pupils were equal. There was a right extensor plantar reflex. B.P. was 130/82, pulse rate was 58/min., and respirations were 18/min. Roentgenograms of the skull showed a linear fracture in the right occipital area traversing the right lateral sinus (Fig. 10).

A diagnosis was made of possible extradural hematoma of the right posterior fossa or a right intracerebellar hematoma with possible contrecoup left temporal or frontal intracerebral clot.

The patient was observed for an hour during which his symptoms became more severe and a markedly irregular bradycardia of 40/min. developed. He was given 100 cc. of 50 per cent glucose intravenously and emergency trephination was scheduled.

Operation. Under general intratracheal anesthesia, with the patient in the prone position in the cerebellar head rest, the laceration of the right occipital region was debrided, and a burr hole was made directly over the linear fracture above the transverse sinus. Upon incision of the dura mater brain bulged through the aperture. A right suboccipital burr hole was next made beneath the transverse sinus and directly over the fracture line, revealing a 3.5 cm. laceration of the dura just medial to the mastoid process. When the dural incision was enlarged necrotic cerebellum

FIG. 10. *Case 4.* Roentgenogram indicating right linear suboccipital fracture.

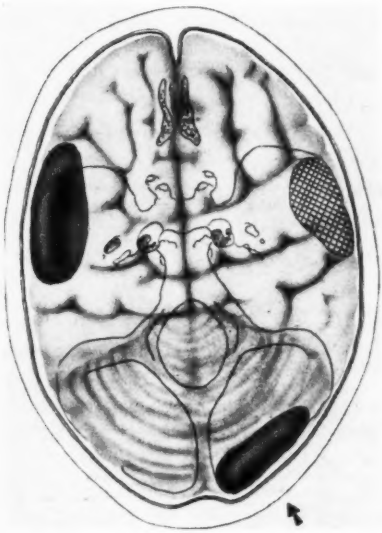

FIG. 11. *Case 4.* Diagrammatic pattern of right intracerebellar hematoma, major left intracerebral clot, and minor right temporal contusion.

and intracerebellar hematoma extruded under markedly increased pressure. Several superficial and deep bleeding points were found and coagulated. The rim of the foramen magnum was removed, the cisterna magna was opened, and the remaining portion of the right cerebellar hemisphere was noted to be without pulsation. The bone was rongeured upward along the fracture line across the right transverse sinus to the burr hole above the sinus to rule out an extradural hematoma. The wound was closed tightly without drainage. Another burr hole was placed in the left suboccipital region, and upon dural incision the nonpulsating brain protruded with no evidence of hematoma.

Because of the evidences of unrelieved pressure, a contrecoup lesion was regarded as most likely. Consequently a left temporal burr hole was made and an acute subdural hematoma and an intracerebral clot were subtotally drained. Since adequate exposure could not be obtained in the prone position, the wound was dressed, and

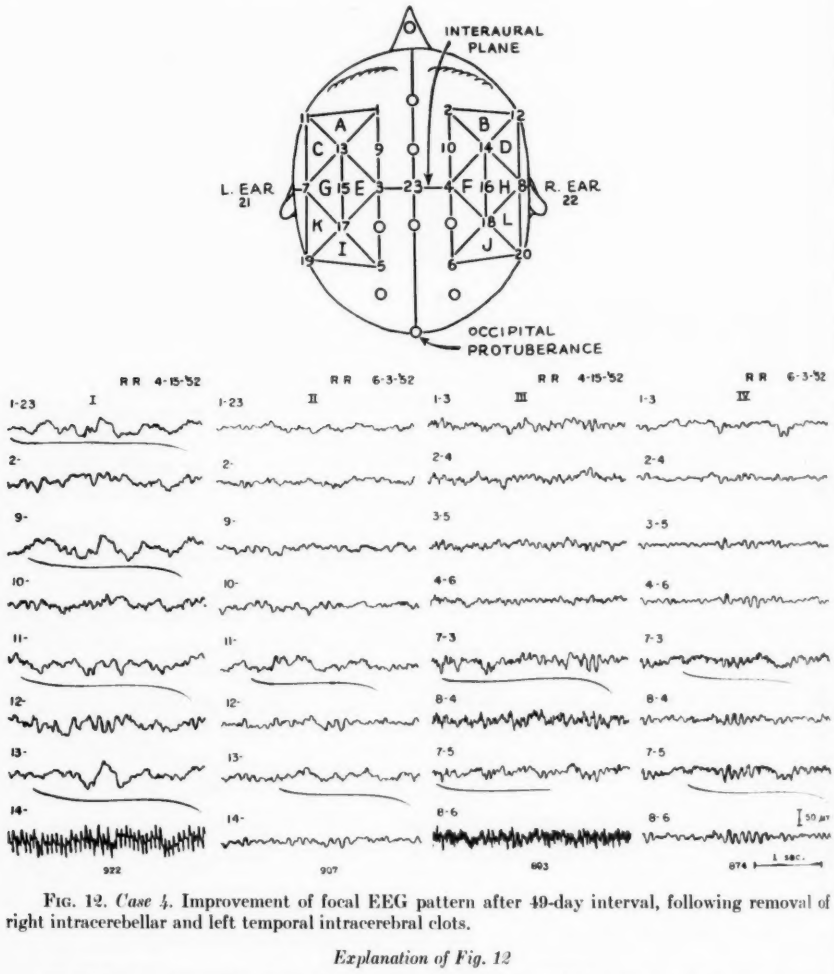

FIG. 12. *Case 4.* Improvement of focal EEG pattern after 49-day interval, following removal of right intracerebellar and left temporal intracerebral clots.

Explanation of Fig. 12

(I) 20 days postoperative. *Strips 1, 3, 5, and 7:* Frequent focal high voltage 1 to 3/sec. abnormal waves in left anterior quadrant with vertex as reference lead. This abnormal pattern may be caused by edema and other effects following surgery.

(II) 69 days postoperative. *Strips 5 and 7:* Focal waves are still present but they are seen much less often and extensive than in I, indicating noticeable improvement. The background has improved too with better establishment of normal alpha pattern and lessening of slow waves (not entirely shown).

(III) Another epoch of recording taken at the same sitting as I: Frequent focal waves in *strips 5 and 7*, left midtemporal region.

(IV) Same epoch of recording as in III but taken at the same sitting as II: Focal waves are still present but less often than in II. There have been some consistent changes in right temporal and neighboring regions and both premotor regions but they are much less now (not shown).

the patient was turned over into the supine position, prepared, and redraped. The burr hole in the left temporal region was enlarged to permit better drainage. After the residual hematoma and a mass of necrotic cerebral tissue 4 cm. in diameter were evacuated the brain pulsated readily. Bleeding was controlled by cautery and tamponade with oxycel cotton. Additional burr holes were made in the left frontal and posterior parietal regions with rubber drains inserted to permit through-and-through drainage. The wounds were then closed. Upon placing a final burr hole in the right temporal area a contused but pulsating brain was found (Fig. 11).

Postoperative Course. The patient could speak and move all four extremities, but had a severe meningeal reaction from subarachnoid bleeding, as manifested by headache, backache, and photophobia. He gradually recovered so that at the time of his discharge 3½ weeks later, although still quite weak, he was able to walk without aid. There was minimal ataxia on right finger-to-nose test and slight nystagmus upon looking to the right.

A follow-up examination on June 9, 1952, showed no neurologic abnormality. EEG studies (Fig. 12) indicated progressive improvement of the focal pattern.

Comment. If the patient had not been operated upon promptly he would have succumbed rapidly from medullary compression. The correct diagnosis of both right intracerebellar hematoma and contrecoup temporal intracerebral clot was considered preoperatively because of our experience with the previous cases.

DIAGNOSIS

The importance of an abrasion or laceration of the scalp in the occipital region with concomitant suture line separation or fracture over the transverse sinus or through the foramen magnum should be re-emphasized. There are many individuals who suffer such injuries and survive without operative intervention. However, in the past 2 years we have seen 15 patients with these objective findings, and in only 7 instances did we feel that the patient could be treated conservatively without exploration. The latter group were alert, had minimal headache, and did not appear acutely ill compared to the other 8, who were extremely sick, suffering from excruciating headache and severe projectile vomiting. In the majority of the cases the state of consciousness varied from lucidity to varying degrees of coma with minimal neurological findings referable to the posterior fossa. In most instances there was little or no change in the vital signs to indicate a rapidly expanding intracranial lesion, and if such a transition is awaited the patient may succumb before operation can be performed. Rowbotham's[22] dictum holds that: "Inspection holes are the only means by which a definite diagnosis can be made of massive hemorrhage not associated with a latent interval. In all cases where diagnosis is in doubt, limited explorations under local anesthesia are indicated." (Patients with posterior fossa injuries may be explored under general intratracheal anesthesia, since the prone position on the operating table prevents accessibility to the patient's airway.)

PATHOLOGY

There is little to be found in the literature regarding traumatic intracerebellar hematoma. In the discussion of traumatic intracerebral hemorrhages Martland and Beling[20] stated that "concussion hemorrhages," i.e., ring-shaped perivascular hemorrhages secondary to trauma, are found chiefly in the corpus callosum, the corpora striata and corona radiata. They emphasized that these hemorrhages were rarely seen below the tentorium cerebelli. Mackenzie,[18] Echols, [5] and Habig[11] each reported cases of patients who had sustained a minor head injury or acute stress 1 week, 5 weeks, and 8 weeks, respectively, before admission to a hospital. All had severe headache, nausea, vomiting, negative skull roentgenograms, and succumbed rather suddenly. At autopsy each was found to have a massive intracerebellar hematoma, the etiology of which was obscure. It is our opinion that all of these cases should probably be classified as "spontaneous intracerebellar hematoma of unknown etiology" since the degree of trauma was apparently negligible in each instance. Hawkins and Rewell[12] and Margolis *et al.*[19] have indicated that if the periphery of this type of hematoma is carefully studied a tiny vascular malformation may be identified as the source for hemorrhage.

Other references may be available on traumatic intracerebellar hematoma, but the only significant one that we have found that satisfactorily describes the lesions presented here is from the pathology literature. Gonzales *et al.*,[7] in describing linear fractures of the occipital bone, stated that they may arise in the posterior portion of the parietal bone and run into the posterior fossa toward the jugular foramen. Others are directed into the middle fossa and some involve the middle ear. Another type begins in the midoccipital region and diverges along the lambdoidal sutures into the right and left middle fossae. The following complications were listed: (1) Contrecoup lacerations are found on the under surfaces and tips of the frontal and temporal lobes, which may give rise to subdural hematomas. These may extend deeply into the subcortical white matter. (2) Direct lacerations are encountered over the cerebellum or on the occipital lobes and are usually minor injuries. "In a few cases, however, the cerebellum may be lacerated and bleed freely into the space below the tentorium causing death by cerebral compression." We believe this injury is more common than was previously realized.

The occurrence of contrecoup lesions has been known since the days of Hippocrates and Galen.[14] However, it has been only within relatively recent years that the general mechanisms of this injury have been investigated. Rowbotham[22] has adequately summarized the ways in which these lesions are produced: "a) by shearing forces; b) by suction, either when the brain is flung from the skull when the motion of the head is abruptly arrested against a resistance, or in distortions of the skull; c) by the brain being struck by a distant arc of the skull as it is flattened in a deformation; and d) by the brain being thrust against a dural septum or face of the skull when

its opposite side is struck by a local inbending of bone—linear acceleration." These injuries of the posterior fossa would fall into categories b) and d).

The hematoma at both sites is caused by laceration of the brain with disruption of the cortical vessels at the surface and to a sufficient depth that the hemorrhage becomes tamponaded subcortically and does not gain complete access to the subarachnoid space. Bleeding then occurs at the expense of the cerebral tissue and if it is in the cerebellum may cause compression of the vital centers in the medulla.

TREATMENT

In most instances the presence of a supratentorial lesion will be quite apparent to the surgeon, and this problem should be treated promptly. However, the rapidity of deterioration of these patients has firmly convinced us of the importance of trephining over the posterior fossa fracture site initially to prevent medullary compression.

The occipital contusion or laceration may be debrided, using intratracheal anesthesia with the patient in the prone position in the cerebellar head rest. A burr hole may first be made in the posterior fossa next to the fracture line, exposing the dura mater and possibly an extradural hematoma. In all of these cases of intracerebellar hematoma there was a 3 to 4 cm. tear in the dura mater through which clot and devitalized cerebellum were extruding under markedly increased pressure. This material must be removed by suction and the dural aperture then be enlarged toward the midline, permitting intracerebellar clot to be evacuated and allowing exposure and cauterization of bleeding vessels. The rim of the foramen magnum is then rongeured away and the cisterna magna opened so that there is a free flow of spinal fluid. The residual necrotic cerebellum is debrided, bleeding controlled, the dura mater left open for decompression purposes, and the remainder of the wound closed solidly in layers. Drains have been omitted from the posterior fossa incisions for fear of permitting a permanent cerebrospinal fluid fistula with the risk of exposure to meningitis or arachnoiditis and their associated complications. Another burr hole is then made in the contralateral suboccipital region to exclude any extradural or intracerebellar lesion; so far none has been found.

McKenzie[17] has pointed out that if an extradural hematoma has been found in the posterior fossa a burr hole should be placed above the transverse sinus to rule out extension of the clot upward to the occipital region. The converse is also true. If an extradural clot is found over the occipital pole, it may have drained downward into the posterior fossa. In all cases of intracerebellar clot we have made routine burr holes above the sinus and have found only evidences of increased intracranial pressure. However, in 2 of our 4 previously reported cases of extradural hematoma of the posterior fossa we have found clots above the transverse sinus, using this routine.

Some neurosurgeons frown upon so-called "woodpecker" surgery, and tend to regard it as an indication of weakness on the surgeon's part. How-

ever, there is probably no other type of intracranial injury that so definitely requires this approach. The improvement in our mortality rate in head injuries has justified it.

After the posterior fossa exploration, if there is continued herniation of the brain at the infratentorial burr holes or if the patient becomes progressively worse there should be no hesitancy in placing additional burr holes in the contralateral temporal and frontal regions to exclude a contrecoup subdural or intracerebral clot. If such a lesion is discovered the burr holes are enlarged, the clot is evacuated, and through-and-through drains are inserted for 24 hours. Antibiotics and the sulfa drugs are administered to prevent infection.

DISCUSSION

The question may well be raised why more extradural hematomas of the posterior fossa and intracerebellar hematomas have not been reported. There may be several reasons why they have not been identified at autopsy. All of these hematomas occur more or less acutely. In the routine postmortem examination in many hospitals the brain is not removed, or if it is, an apathetic prosector frequently does not bother to strip the dura mater from the skull, thereby failing to expose an extradural lesion. In most institutions where intracranial postmortem examinations are made the calvarium is opened and the brain is removed by cutting across the falx and the tentorium with incision of the venous sinuses and drainage of blood into the posterior fossa. A fresh traumatic hematoma in this region may be confused with this apparently artefactual collection. Finally, if severely contused and softened brain is removed, the prosector's fingers often tend to slip into the frontal and cerebellar hemispheres as the brain is withdrawn so that any cerebellar laceration or intracerebellar clot may be regarded as partially artefactual. The general observers viewing such a specimen at autopsy are frequently heard to comment: "He did not have a chance with that amount of brain damage." This is not true, for 3 of our 4 patients with these lesions survived, and 2 of them are quite normal individuals.

More of these lesions will be found at the operating table in the future if they are carefully sought, and if aggressively treated, their prognosis is relatively good.

SUMMARY

Four cases of intracerebellar hematoma with associated contrecoup lesions are reported in detail. In 3 of these, contrecoup intracerebral clots were found in the temporal and frontal regions, and in the remaining case severe contusion was diagnosed by burr holes, neurologic findings and EEG tracings. The postoperative and/or follow-up EEGs are presented and discussed. Preoperative ones could not be made, because of the rapid pathogenesis of the lesion.

In any severely injured patient who has a scalp contusion or laceration over the occiput, a suture line separation or fracture line traversing the

lateral sinus or extending through the foramen magnum, trephination of the posterior fossa is justified. In such a case even if a posterior fossa lesion is found the surgeon must be constantly on the alert for contrecoup intracerebral hematoma in the temporal or frontal region and be prepared to evacuate it.

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PARALYSIS OF THE ULNAR NERVE AND MANAGEMENT OF ITS DEFORMITY

WITH SUGGESTION FOR TEMPORARY PARTIAL BLOCKAGE DURING RECOVERY

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PARALYSIS of the ulnar nerve has been more frequent and unfortunately the results of suture of this nerve have given less gratifying results than in any other peripheral nerve. Reasons for this are: (1) the ulnar is a mixed motor and sensory nerve carrying different types of nerve fibers, (2) distal innervation is to small highly specialized muscles for the finer movements of the hand, (3) another reason may be the poor blood supply of the nerve which may be further impaired by anterior transplantation of the nerve.

The functional return of a useful hand should be the objective when confronted with an ulnar nerve paralysis. This may be obtained by neurorrhaphy or by a combination of primary nerve repair followed by tendon transfers for deficient regeneration. In either case we must preserve the parts of the hand that are essential for function. An innervated, but stiff hand is useless. Steps must be taken to preserve muscles, joints and soft tissues in a state of mobility.

It seems to the writer that mechanical splintage of paralyzed joints must follow more closely the anatomical and physiological characteristics of a part. The prevention of muscle stretching is important, but the joint structures on which the muscle acts must also be considered; namely, the ligaments, joint capsules and soft tissues. For example, tight skin about a joint can vitiate completely normal nerve-muscle-joint mechanism.

Paralysis of the ulnar nerve sparing the nerves to the flexor digitorum profundus muscle presents a deformity of the hand that is not seen when the lesion is above the elbow. Phylogenetically, the muscles innervated by the ulnar nerve are oldest and more highly co-ordinated than those supplied by other peripheral nerves. This nerve is peculiar by supplying muscles at different levels in the extremity, but which co-ordinate for fine hand movement involving identical joints. An active flexor digitorum profundus muscle sets in motion the platform of origin of the lumbrical muscles. The latter, however, have their insertion upon the dorsal expansion of the metacarpophalangeal joint.

Paralysis of the ulnar nerve above the elbow, producing a flail state of all the muscles supplied by this nerve, causes a minimal deformity of the hand. However, a lesion below the elbow sparing the innervation of the

flexor digitorum profundus produces an unequal pull on the "a" joint of the little and ring fingers. The deep flexors to these digits flex the interphalangeal joints. The lumbrical muscles initiate the arc of flexion in the "a" joints. When the latter are paralyzed, this joint is tipped into extension by the extensor tendon. This position is maintained by the continuous pull of the active flexor digitorum profundus muscle. Apparently, an active

FIG. 1. Typical typewriter-deformity of the hand seen in ulnar nerve paralysis. The deformity is usually more severe when the innervation to the flexor digitorum profundus muscle is spared.

lumbrical muscle is the key to a neutral balance between extension and flexion of the metacarpophalangeal joint. Though less noticeable, the same pattern of deformity is present in median nerve paralysis if the lesion is distal to the innervation of the deep flexor muscles to digits 2 and 3. If the median nerve alone is paralyzed, the intact interossei help maintain the balance. However, a combined ulnar and median nerve paralysis gives rise to the claw hand. This deformity is produced by the same mechanism.

An obvious and interesting fact of ulnar nerve paralysis is that the deformity associated with lesions below the elbow is greater than that with

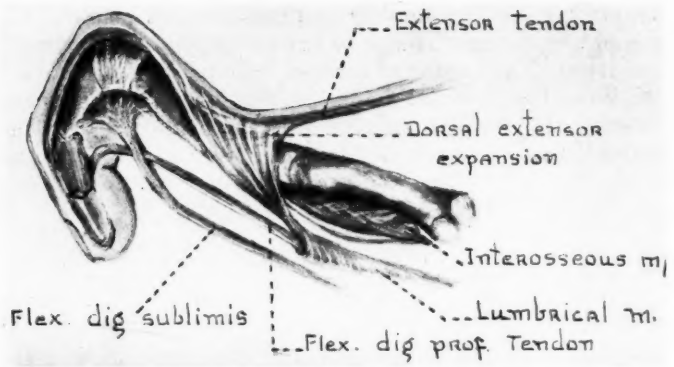


FIG. 2. The lateral view of the deformity shows the dorsal extensor apparatus of the "a" joint pulled upwards by the active extensor muscle. The paralyzed lumbrical muscle has allowed the proximal phalanx to tip into extension. The flexor digitorum profundus muscle pulls the distal phalanges into flexion.

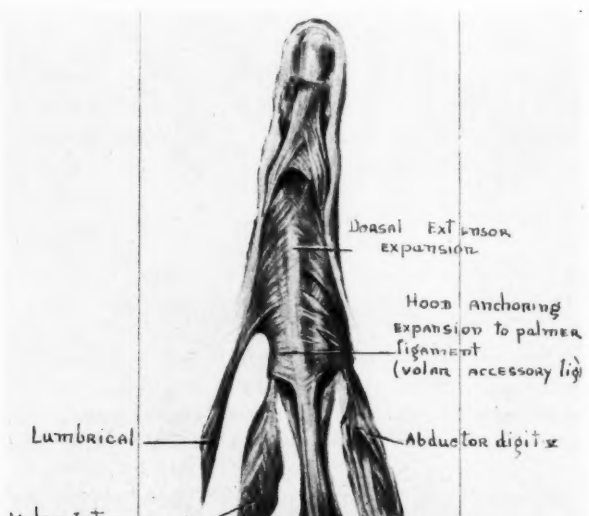
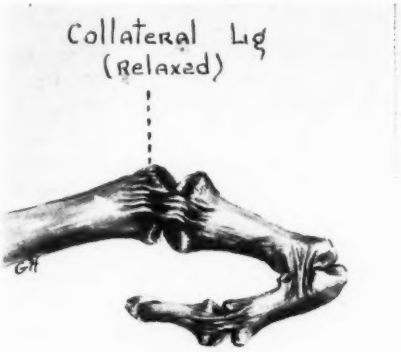


FIG. 3. The dorsal view of the extensor apparatus reveals the lumbrical and volar interosseous muscles inserted to the dorsal hood. The lumbrical muscle originates on the flexor tendon but is inserted on the dorsal expansion. In the little finger the latter muscles are inserted on the radial side. The abductor digit quinti substitutes for the interossei on the ulnar side. (Redrawn from Grant)[3].

lesions above the elbow region. This results from the muscle imbalance exerted on the hand when the innervation to the ulnar portion of the profundi flexor is intact. This observation has been recorded by Bunnell[1] and by Davis.[2] When this muscle is activated, a flexion contracture is superimposed upon the little and ring fingers. This accentuates the hyperextension deformity of the "a" joint because the paralysis of the interossei and lumbrical muscles, which extend joints "b" and "c" and flex the "a" joint, allows the unopposed action of the extensor tendons to throw the



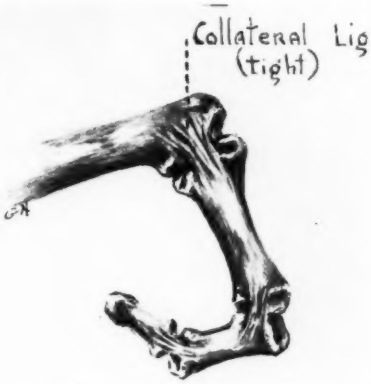


FIG. 4 (*left*). The ligaments of the metacarpophalangeal joint are relatively relaxed when the corresponding joint is in the neutral position. This allows adduction and abduction in the joint.

FIG. 5 (*right*). The collateral ligament of the metacarpophalangeal joint is tense when the joint is in acute flexion. This prevents adduction and abduction motion in the joint.

proximal phalanx into extension. This attitude of the digit allows the middle and distal phalanges to fall into flexion; only however, if the flexor tendons are active.

The physiological effect of this uneven muscle pull manifests itself early by deformity of digits 4 and 5, especially so in the latter, described as the typewriter position of the fingers, and later by limitation of motion in "a" joints. The limitation of motion in the "a" joint is caused by thickening and shortening of the collateral ligaments of the joint and peri-articular fibrosis at the interphalangeal joints. This effect apparently is not from disuse, but from the uneven pull exerted about the joint by the tendons involved. Clinically, this is obvious in brachial plexus lesions when the small joints of the hand are flail from a complete paralysis in the extremity. No motion is possible in the joints except passively, but they remain supple even months after injury.

When the ulnar nerve lesion is above the elbow, the patient retains the ability to clench a fist though there is little power in the little finger. If the flexor digitorum profundus is paralyzed the ugly deformity of the hand is absent, and with the passage of time incident to nerve regeneration following

suture the "a" joint maintains its mobility and function until the deep flexor is re-innervated. As the flexor muscle gains power, the deformity becomes more evident.

Anatomically the metacarpophalangeal joint is supported by two pairs of ligaments: the volar and the dorsal; and the two collateral ligaments. The collateral ligaments have fibers running parallel to the joint surface. When the "a" joint is in extension the collateral ligaments loosen up and allow abduction and adduction of the joint. When the joint is in complete flexion, the collateral ligaments shorten and prevent abduction and adduction. Therefore, the latter motions are impossible when the finger is in flexion, whether this is caused by muscle imbalance or because the finger is kept in flexion by external splintage. It seems that the usual type of splint for this deformity directs its force to further tighten the collateral ligaments, by increasing the flexion of the joint and eliminating abduction and adduction movements. The volar ligaments do not seem to hypertrophy as much as the collateral, probably because the line of directional stress upon the ligament is different.

FIG. 6. Paralysis of the ulnar nerve above the elbow. This shows cupping of the palm from atrophy of interosseous muscles. Note that this man can extend completely digits 4 and 5. There is no tension on the capsule of the metacarpophalangeal joint.

It has been noted on various occasions following anterior transplantation of the ulnar nerve necessary for nerve suture that for several days following surgery the 4th and 5th digit deformity in the hand disappears and the patient is relieved from the constant tension on these digits. He feels better! This is probably secondary to a temporary paralysis of the fibers to the deep flexors from handling the nerve fibers in dissecting them from the main nerve trunk and from edema in the surgical wound. The power to make a fist does not seem to be impaired. The decreased deformity has also been noted in high lesions of the ulnar nerve; this becoming worse as the nerve fibers regenerate to activate the deep flexor muscles. On several occasions, the neuroma-in-continuity involved sacrificing the motor branches to the flexor profundi muscle before normal-appearing fascicles could be obtained for nerve suture. In these cases also there was a disappearance of the deformity and each patient presented a better hand. These observations suggest the benefit that may accrue from a partial temporary blockage of the nerve in treatment.

The muscle imbalance in low ulnar nerve lesions could be temporarily

FIG. 7. Paralysis of the ulnar nerve below the elbow. The little finger shows the typical typewriter attitude with the metacarpophalangeal joint hyperextended and the finger in flexion.

eliminated by dissecting free the innervation to the flexor digitorum profundus from the main nerve trunk, a sufficient distance proximal so that the fibers could be crushed much in the fashion that a phrenic nerve is, to produce a temporary paralysis of this muscle. If this were done from 10 to 12 cm. above the myoneural junction the tension effect of this muscle upon the metacarpophalangeal joint producing thickening and deformity of the small joints in digits 4 and 5 would be postponed sufficiently long to allow innervation of the distal muscles, namely, the interossei and the lumbricals. We have not as yet performed this procedure but there is no reason to believe that a permanent paralysis of the deep flexor would result from crushing its nerve supply; however, even if it did the ability to make a fist would not be impaired.

FIG. 8. This man had severance of the ulnar nerve above the elbow. He can make a good fist without any deformity.

We have performed several amputations of the little finger with its metacarpal head because of deformity secondary to irreparable lesions of the ulnar nerve below the elbow. This was necessary because the metacarpophalangeal joint was stiff from fibrosis of its capsule. These patients had

routine physiotherapy consisting of passive exercises of the finger joints, hot bakes, massage, hot paraffin treatment and electrical stimulation. In one of these irreparable lesions the sensory component arose from the the nerve trunk at a higher level than the penetrating wound. This man had sensation over the ulnar distribution in the hand. If the small joints could be saved in a case like the above, the patient would still have a good functioning hand or one amenable to tendon transplants; assuming, of course, that the nerve suture had failed completely. This could have been accomplished by defunctionalizing the ulnar half of the flexor digitorum profundus muscle. This procedure seems like destructive surgery, but in the long run would have proven to be conservative therapy eliminating amputation of the 5th digit and its corresponding distal third of the metacarpal bone and would have resulted in a useful hand without deformity.

SUMMARY

1. Paralysis of the ulnar nerve below the elbow gives more deformity of the hand than paralysis above the innervation of the flexor digitorum profundus muscle.

2. Constant flexion of the 5th metacarpophalangeal joint produces fibrosis of its capsule with ankylosis of this joint.

3. Defunctionalizing the flexor digitorum muscle would diminish this fibrosis and deformity of the 5th metacarpophalangeal joint. This could be done by crushing the motor fibers to this muscle.

4. Splinting the 4th and 5th digits in flexion for ulnar nerve paralysis may be unphysiological.

5. Temporary denervation of the deep flexors to digits 2 and 3 from median nerve paralysis alone or accompanied by ulnar paresis would minimize *main en griffe* or claw hand.

6. Prevention of stiff hand joints by eliminating the factor of tension gives the constructive surgeon a better chance to effect tendon transfers. Hands requiring tendon transfers must be supple and pliable.

7. An innervated but stiff hand is useless.

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THE MYELOGRAPHIC APPEARANCE OF ADHESIVE SPINAL ARACHNOIDITIS

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A REVIEW of the literature reveals a paucity of illustrations of the myelographic appearance of spinal adhesive arachnoiditis.[1,3,5,6,11,12] Most of those published are of the extensive variety that cause little diagnostic difficulty. In view of the recent report of Kennedy *et al.*[8] emphasizing the frequency of adhesive arachnoiditis following spinal anesthesia, it was felt that publication of the following 7 surgically verified cases would be of interest. The clinical features and therapeutic aspects have been recently reviewed by one of us,[10] and the present communication will be limited to the radiographic findings in verified cases.

Since the diagnosis of chronic adhesive spinal arachnoiditis is frequently a difficult one to establish clinically, myelography may be a very helpful procedure. In 1929 Odin and Runström[9] reported 14 cases of arachnoiditis and described the myelographic picture as one showing the contrast material in the form of droplets and streaks. From a study of the pathologic alterations produced by arachnoiditis, they postulated that the following alterations might occur in the myelographic appearance: a complete block to the passage of contrast material, the formation of pockets with retention of opaque substance, or the production of a filling defect in the contrast shadow. They observed no instances of complete block and most of their cases exhibited the picture characterized by streaks and droplets of contrast material.

It was probably because of these multiple possibilities that Dyke[3] wrote that there is no characteristic picture of this condition. He mentioned complete and incomplete obstructions to the passage of opaque material as occurring in this disease as well as scattering of opaque oil in small globules over a large portion of the spinal canal. Davidoff, Gass and Grossman[2] pointed out that a localized adhesive spinal arachnoiditis could occur following the removal of a cord tumor and simulate a recurrence by both clinical symptoms and myelographic findings. In 3 of their 5 cases a complete block to the flow of opaque oil during myelography was demonstrated.

Elkington,[4] in a recent review of arachnoiditis, stated that myelography is perhaps the most valuable aid to the correct diagnosis. In his experience a clear-cut arrest of the contrast medium either above or below the affected segments of the cord is rare. He stated that in some cases the picture re-

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sembles the guttering of a candle or of a sheath lining the circumference of the spinal canal in a manner difficult to explain other than by a diffuse and partial occlusion of the subarachnoid space.

CASE REPORTS

Case 1. H.W., a 17-year-old colored gravida 1 para 1, was delivered of an apparently normal child on March 28, 1950 under saddle-block anesthesia (2.5 mg. "heavy nupercaine"). The delivery was uneventful as was the anesthetic, except that the patient noted numbness of her body from the rib margin down in spite of the so-called "saddle" technique.

FIG. 1. *Case 1.* Opaque material was injected at the L1–L2 interspace and would not move up or down. Note irregular shape and eccentric position within the spinal canal.

The postpartum period was entirely normal until the 5th week, when she noted slight numbness and weakness in her feet. This condition progressed in severity and on July 3, 1950 she returned complaining of numbness from the waist downward, inability to walk without support, and dysuria for the previous 2–3 weeks.

She was admitted to Barnes Hospital, with clinical findings typical of transverse myelitis at the level of the 7th thoracic dermatome and profound sensory changes below the iliopsoas group. There were bilateral pathological toe signs, and ankle and patellar clonus. Routine laboratory findings and x-rays of the spine were normal. Lumbar puncture was unsuccessful on three attempts.

Myelography. A small amount of pantopaque was introduced into the spinal canal at the level of the 1st and 2nd lumbar vertebrae. This remained stationary and could not be moved up or down (Fig. 1). One cc. of pantopaque was then introduced via cisternal puncture and it moved downward freely to the level of the 8th dorsal vertebra where it met a complete block.

Laminectomy. The dura mater was thickened and firmly adherent to the pia-arachnoid. On incision of the dura mater, the entire spinal cord in the exposed area was found to be surrounded by a dense mass of thickened adherent arachnoid, which could not be dissected free from the pia and cord. A biopsy was taken which confirmed the gross appearance of chronic inflammation of the meninges.

Case 2. F.L., a 29-year-old white gravida 3 para 3, was delivered of a normal child on Jan. 3, 1949 under saddle-block anesthesia (heavy nupercaine 3.75 mg.). The delivery was uncomplicated and the anesthetic was classical in the distribution of hypalgesia with complete recovery in 2 hours. She remained well for 9 months, but in September 1949 she noted numbness in both thighs and legs, slightly more

on the right. There was slow progression and by June 1950 the numbness extended to the "saddle area" and was associated with slight weakness of both legs and an unsteady gait. She had also noted marked hesitancy in voiding and on at least one occasion incontinence of urine.

She was admitted to Barnes Hospital on July 2, 1950. She was an obese white female who walked slowly and unsteadily. Examination revealed a sensory deficit below L3, a diminished right knee jerk, absent ankle jerks, no plantar response, and bilateral weakness of both gluteal groups and anterior tibial groups. There was moderate anal relaxation. Laboratory findings and spine films were normal. Lumbar

FIG. 2. *Case 2.* There is a complete block to cephalad flow at the level of D12. Note the irregular oblique character of the advancing opaque column and the smooth filling defect below suggestive of a tumor.

puncture was unsuccessful below L1, but at that level the CSF dynamics and chemistry were normal. Cystometrogram showed a hypotonic bladder with residual urine.

Myelography. There was a complete block at the level of the lower border of D11, with a large, smooth filling defect at the level of L1 on the right side (Fig. 2). This combination of findings was interpreted as indicating the presence of an intraspinal tumor.

Laminectomy. The dura mater was thickened and adherent to the pia-arachnoid, although it was possible to free some of the adhesions. No evidence of a tumor was found. Biopsy showed chronic inflammation of the meninges.

Case 3. M.A., a 40-year-old white gravida 1 para 1, was delivered of a normal infant on Mar. 1, 1950 under saddle-block anesthesia (heavy nupercaine 2.5 mg.) which produced satisfactory hypalgesia up to the rib margin. The postpartum period was normal until the 3rd month when she noted the onset of intermittent headache and back pain. After 1 month these symptoms disappeared but were replaced by progressive numbness which within 2 weeks had involved the previous area of

anesthesia. Associated with the numbness there was progressive motor loss in both legs until the patient was unable to walk without assistance. She had no bowel or bladder difficulties.

She was admitted to Barnes Hospital on July 3, 1950. Examination revealed a thin white female who walked only with assistance and then showed a slow spastic gait. Routine laboratory findings and spine films were normal. There was a marked sensory deficit below D7. Abdominal reflexes were absent, knee and ankle jerks were hyperactive but equal, and patellar and ankle clonus and pathological toe signs were present bilaterally. There was marked motor weakness in all muscle groups of both

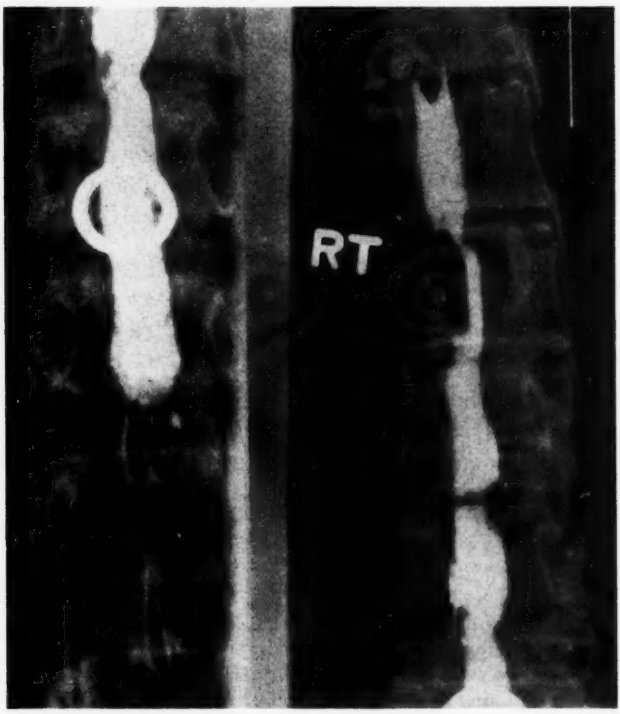

FIG. 3. *Case 3.* Following introduction of pantopaque at the L1–L2 interspace, a complete obstruction to cephalad flow was demonstrated at the level of D7. The opaque column is irregular and there is a filling defect at the level of D8.

lower extremities. A spinal puncture was performed at the L1–L2 interspace, and dark yellow spinal fluid containing 590 mg. protein was obtained.

Myelography. The spinal canal was narrow and irregular in the upper lumbar and lower dorsal area, although the lower lumbar region was within the normal limits. A complete block was demonstrated at the level of D7-D6 intervertebral space (Fig. 3). Because of the large area involved by the pathologic process it was felt to be most compatible with a diagnosis of arachnoiditis.

Laminectomy was performed between D6 and D8. The dura mater was found to be thickened and adherent to the pia-arachnoid and appeared to be strangulating the spinal cord.

Case 4. B.M., a 27-year-old gravida 1 para 1, was delivered of a normal infant April 28, 1950 under saddle-block anesthesia (1 cc. 3 per cent metycaine). The delivery was not remarkable nor was her postpartum course. She was quite well until June 1951 when she first noted that her legs felt somewhat heavy and she was beginning to be uncertain of her balance. She also noted that the floor seemed to feel hot to her. These symptoms progressed until her admission to Barnes Hospital, Dec. 11, 1951.

On admission her gait was unsteady and she swayed markedly in Romberg's position. The quadriceps were weak bilaterally, more marked on the left side. There was also weakness of the muscles of the leg and foot on the left with associated left foot drop. The deep tendon reflexes were slightly hypoactive but equal on both sides. The abdominal reflexes were absent bilaterally and there was no response to plantar stimulation. Sensory examination revealed patches of absent to diminished sensitivity to pin prick, heat and touch, extending from about the level of the 8th dorsal dermatome to the 5th lumbar bilaterally. There appeared to be diminution of vibratory sense over the iliac crests and the patellae of fairly definite degree. Position sense in the lower extremities was intact.

FIG. 4. *Case 4.* Complete block at the level of D12 with demonstration of a large, smooth filling defect suggestive of the presence of an intraspinal tumor.

Laboratory findings were not remarkable. Lumbar puncture revealed an initial pressure of 135 mm. spinal fluid. The Cone test demonstrated an almost complete block. The CSF protein was 197 mg. per cent with a negative Wassermann and colloidal gold curve.

Myelography. The spinal canal appeared normal in the lower lumbar region. Opposite the interspace between the 1st and 2nd lumbar vertebrae a partial block and a smooth oval filling defect were noted (Fig. 4). Some of the opaque material flowed as high as the 12th dorsal vertebra around the right side of the filling defect but no significant amount passed cephalad to this level.

Laminectomy. Because of the possibility of an intraspinal neoplasm as suggested by the presence of a filling defect on the myelogram, it was felt that an exploratory operation was warranted. On exposing the spinal cord the diagnosis of adhesive arachnoiditis was obvious and no evidence of a tumor was found.

Case 5. V.H., a 40-year-old white female, had had four previous hospital admissions for the excision of multiple neurofibromata involving the brachial plexus, intercostal, sciatic and ilio-inguinal nerves on the right side. There was no history of spinal anesthesia. During her fourth hospital stay *myelography* had been performed with the demonstration of an irregular opaque column in the region of the 5th lum-

bar vertebra, a filling defect on the left and a smaller one on the right (Fig. 5). These were thought to represent either multiple neurofibromata or a ruptured nucleus pulposus.

In January 1952 she was admitted to the hospital for the fifth time because of pain in the right leg of 18 months' duration. Examination revealed a diminished right knee jerk and an increased right ankle jerk. Minimal hypalgesia of the lateral two toes of the right foot and right upper outer thigh was present.

Myelography was repeated with findings that were identical to those of the previous examination 7 months before.

Laminectomy. An exploratory operation was performed and the surgeon described thickened, reddish arachnoid with partial obliteration of the subarachnoid space. Just below the left 5th lumbar nerve root was a heavy adhesive band, densely adherent to the arachnoid and dura mater, which was felt to be responsible for the localized myelographic defect seen at this level. No evidence of tumor was found. Microscopically the biopsy specimens showed evidence of chronic inflammation.

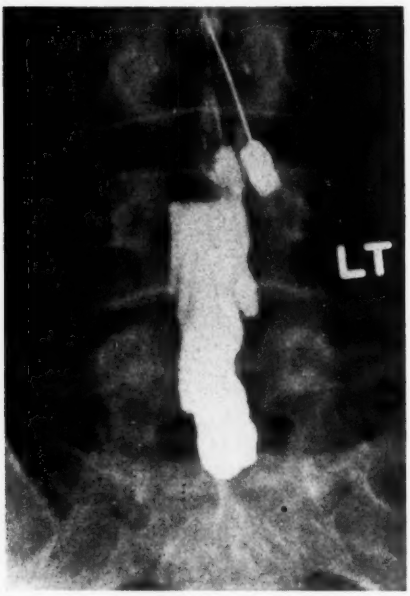

FIG. 5. *Case 5.* Myelogram of a patient with neurofibromatosis. The opaque column has an irregular contour with filling defects on the right and left suggesting multiple tumors. Archnoiditis was found at surgery with no evidence of tumor.

Case 6. B.J., a 36-year-old white male, was admitted to Barnes Hospital in January 1952 for treatment of a urinary tract infection. Ten years before he had experienced a 2-week episode of painful paraplegia with complete recovery. Five years later he began to note gradual progression of weakness and sensory loss of both legs. For the last 2 years the paraplegia had been complete.

Examination disclosed a well developed young male who was paralyzed and anesthetic from the level of the 9th dorsal nerve down bilaterally.

Myelography. The spinal canal was irregular in both the lumbar and lower dorsal region with a complete block at the level of D9. At the level of T12 a 1×2 cm. oval irregular filling defect was demonstrated that suggested the presence of a tumor (Fig. 6). Although the appearance of the myelogram suggested arachnoiditis, it was felt that because of the presence of the filling defect a laminectomy was warranted to exclude the possibility of an associated spinal neoplasm.

Laminectomy. On Jan. 2, 1952 an exploratory operation was done, with removal of the 8th and 9th dorsal laminae. The subarachnoid space was obliterated by thickened, opaque fibrotic arachnoid and the spinal cord appeared atrophic. No evidence of tumor was found.

Case 7. J.G., a 48-year-old negro, entered Barnes Hospital because of weakness

of both lower extremities of 2 years' duration coming on following an acute illness characterized by fever, chills and stiff neck. He also complained of impotence and urinary incontinence.

Examination revealed a markedly spastic gait with hyperreflexia and Babinski responses in both lower extremities. Spinal puncture and associated manometric tests disclosed a partial subarachnoid block. The CSF protein was 77 and 83 mg. per cent on two occasions but the Wassermann and colloidal gold were negative.

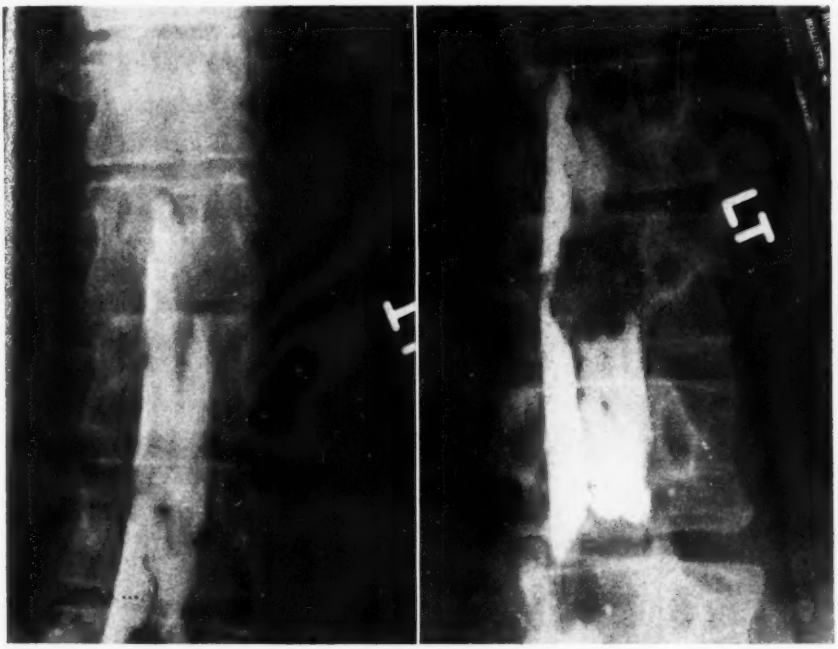

FIG. 6. *Case 6.* (*Left*) Complete block at the level of D9 with an irregular frayed margin at the head of the opaque column. (*Right*) Apparent filling defect suggestive of the presence of an intraspinal tumor.

Myelography. The opaque oil was observed to flow normally to the level of the interspace between D10 and D11 where a complete block was found. The leading edge of the opaque column had an irregular frayed appearance. A small amount of opaque oil was then introduced by cisternal puncture and a complete block was found at the D1 and D2 interspace. Because of the extent of the block, from D2 to D10, it was thought that arachnoiditis was the most likely diagnosis (Fig. 7).

Laminectomy. An exploratory operation was carried out and the arachnoid membranes were found to be gray, opaque and dense. Several small loculations of spinal fluid were seen as well as adhesions binding down some of the dorsal roots.

DISCUSSION

The deviations from the normal myelogram observed in the above-described cases of spinal adhesive arachnoiditis coincide with what one

might predict from the gross pathological changes found in this disease. When obliteration of the subarachnoid space is total, a complete block is found on myelography. If obliteration is partial or patchy, filling defects and/or pockets are demonstrated by myelography. These appearances may be indistinguishable from the changes produced by either tumors or ruptured intervertebral discs.[9]

Jantz[7] has emphasized the difficulty of differentiating spinal cord tumor

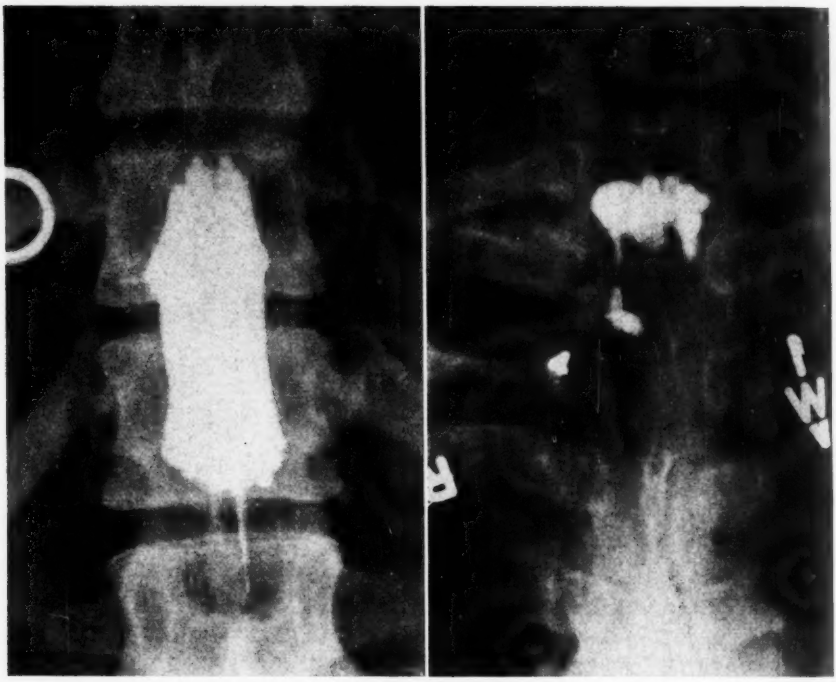

FIG. 7. *Case 7.* (*Left*) Complete block at level of upper border of the body of D11. Head of the opaque column has an irregular, ragged edge. (*Right*) Opaque oil introduced via cisternal puncture demonstrates the upper border of the obliterated arachnoid space at the level of D1–D2 intervertebral space.

and spinal arachnoiditis when there is a complete block. He states that in the presence of either an intra- or extradural tumor when a complete block occurs the margin of the opaque column adjacent to the block is smooth and homogeneous. In the presence of arachnoiditis it is ragged or irregular because of adhesions that extend beyond the point of the obstruction. This observation was confirmed in 4 of our cases (Figs. 1, 3, 6 and 7) and may be a helpful sign. However, in 4 patients (Figs. 2, 3, 4 and 6) the presence of an intraspinal tumor was suspected because of a large, smooth filling defect. Presumably these represented localized areas of complete obliteration of the subarachnoid space.

The classical picture of extensive arachnoiditis in which the contrast material is spread out in the form of streaks and droplets may be readily recognized although it might be confused with subdural and extradural injections of radiopaque oil. Actually the roentgen picture of adhesive arachnoiditis is quite different from that of misplaced oil. In 3 cases we were able to demonstrate obliteration of a large area of the subarachnoid space, and a diagnosis of arachnoiditis was considered likely despite the absence of the so-called "characteristic" myelographic picture. The coexistence of arachnoiditis with other lesions must always be considered. Localized arachnoiditis has been reported associated with ruptured intervertebral discs.[5]

SUMMARY

1. The myelographic appearance of 7 surgically proved cases of adhesive spinal arachnoiditis has been presented.

2. The myelographic changes produced by chronic adhesive arachnoiditis consist of complete or partial block, formation of pockets and filling defects, and the spreading out of the opaque oil in the form of streaks and droplets.

3. Because of the occurrence of a complete obstruction and filling defects, differentiation from intra- and extradural tumors is difficult. An irregular ragged margin of the opaque column at the site of obstruction favors the diagnosis of arachnoiditis.

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THE TREATMENT OF RUPTURED LUMBAR INTERVERTEBRAL DISCS BY VERTEBRAL BODY FUSION

I. INDICATIONS, OPERATIVE TECHNIQUE, AFTER CARE

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Whenever the subject of the treatment of ruptured lumbar intervertebral discs is discussed, one always finds controversy and disagreement among neurosurgeons and orthopedic surgeons on the advisability, indication and necessity of a spinal fusion in these cases.[2] The reason for this controversy is obviously the fact that many patients who have had operations for the removal of a ruptured intervertebral disc have not been cured of their symptoms or have returned at a later date with a recurrence of them.

Dandy[12] stated very positively that spinal fusions were absolutely unnecessary and that one had only to curette out the nucleus pulposus and the involved joint would fuse itself! More conservative neurosurgeons[14,16,19,21,24,27] believe that if a patient has leg pain predominantly, simple removal of the disc may be sufficient. When the disability is primarily low-back pain and the patient has abnormal facets or arthritic spur formation giving evidence of an unstable back,[1,25] a fusion operation is indicated. Most orthopedic surgeons are in favor of combining the removal of the herniated portion of the ruptured disc with a fusion operation.[13,15] In the light of personal experience in the surgical treatment of ruptured lumbar discs since 1937 and in studying the results of others, it is my opinion that more patients will be permanently cured of their symptoms if removal of the ruptured disc is accompanied by bony fixation of the involved vertebral joint. The writer is one of few neurosurgeons who seriously believes that all lumbar disc operations should be accompanied by a spinal fusion, if the fusion is done between the bodies of the vertebrae.

The two types of disabling pain associated with ruptured intervertebral discs are produced by various pathological factors. (1) The *low-back pain* is primarily caused by collapse of the intervertebral disc and the resultant loose, unstable vertebral joint. The torn fragments of the disc may move about within the intervertebral space. If loose fragments shift to one side of the interspace, they may interfere with the normal articulation of one vertebral body upon the other, producing changes of alignment of the spine, limitation of movement and pain. There may result from this situation muscle spasm, which will also contribute to the back pain. Overriding of the articular facet resulting from collapse of the disc causes traction upon the joint capsule, which is painful.

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(2) The *pain in the lower extremities* may occur with compression of the nerve root at the intervertebral foramen. One or all of the following abnormal conditions may be responsible for nerve root pressure: (a) Herniation of the torn fragments of the intervertebral disc or bulging backward of the nucleus pulposus into the spinal canal. (b) Narrowing of the intervertebral foramen by subluxation of the articulate facets following collapse and narrowing of the intervertebral space. (c) Proliferation of bone along the margins of the body of the vertebrae posteriorly, (osteophytes) encroaching upon the intervertebral foramen. (d) Hypertrophy of the ligamentum flavum which, with the facet, forms the posterior boundary of the intervertebral foramen. This latter condition, however, is questioned by some writers.

In the medical literature of the past 15 years on the treatment of ruptured intervertebral disc, most emphasis has been placed upon the relief of the sciatic pain by removal of the mechanical pressure upon the nerve root.[27] The primary pathology, which is a damaged or broken joint of the spine, has received less attention. In recent years, however, more and more spinal fusion operations are being advocated, indicating a trend toward treatment of the joint pathology.[1] Obviously both causes of disability must be considered and treated if one expects to completely cure the patient of all symptoms. First, one must remove all abnormal mechanical factors that may compress the nerve root; and second, one must immobilize the broken, movable, painful vertebral joint. Removing the herniated fragments of the intervertebral disc without immobilizing the joint disregards the primary pathology and, likewise, fusion of the spine without thorough investigation and decompression of the involved nerve root cannot be expected to relieve the patient of the radiating pain in the lower extremities.

In 1944, after the appearance of a report by Shinners and Hamby,[23] the writer made a follow-up survey of 162 patients who had been operated upon for ruptured disc in the previous 5-year period. They had all had hemilaminectomies with simple removal of the protruded disc. The number of patients who had continued to complain of low-back pain or recurrence of sciatica was impressive. Over 70 per cent of them had returned to the office at one time or another after operation with complaints referable to their ruptured disc. [Of these patients 32 or 19.7 per cent subsequently had a second (fusion) operation.] It was concluded that any operative procedure that could not promise the patient at least 50 per cent chance of a complete and permanent cure was not worth while. The writer therefore set about to devise some procedure that could permanently cure these patients without disability. It seemed logical that to correct the underlying cause of the low-back pain, i.e., the collapse of the intervertebral disc, an operative procedure should be directed toward immobilizing the abnormal vertebral joint.

A series of patients were operated upon with an orthopedic surgeon. A posterior (Hibbs) type of spinal fusion was done after removal of the herniated fragment of disc. We were not satisfied with our results. It never

seemed physiologically correct to me to attempt to immobilize a single vertebral joint; for example L4 and L5, by bridging a graft over the spinous processes and laminae (non-weight-bearing surfaces) of the joint above and the joint below. This type of fixation operation gave a stiff back, which was very often painful, and failure of fusion (pseudo-arthrosis) occurred all too frequently. We had the same experiences as Love;[17] 2 of our patients had a recurrent herniation of the same disc with return of the sciatica after a posterior spinal fusion had been done, and in 3 patients a ruptured disc developed above a spinal fusion.

The ideal operation, mechanically efficient, for the fixation of a single vertebral joint after rupture of an intervertebral disc should be one directed toward restoring the intervertebral space, if possible, and at the same time immobilizing the adjacent vertebral bodies. Fusion of the chief weight-bearing part of the vertebra—the bodies—by a strong intervertebral graft wedged into the interspace to replace the ruptured disc seemed to be the logical procedure. The operation of vertebral body fusion was begun in 1943, and almost every patient with ruptured lumbar disc operated upon since 1944 has been treated in this manner.

The author was not aware then of the experience of Van Wagenen[28] who had attempted a similar procedure at about the same time but had discontinued it. Serious complications had resulted when the intervertebral graft had worked loose and herniated posteriorly into the spinal canal, compressing the cauda equina. The following year (1944) Briggs and Milligan[3] reported having packed the intervertebral space with bone chips in an effort to obtain an intracorpus fusion. These authors also recommended driving a round peg of bone into the interspace for stability. In 1945 Ovens and Williams[20] reported a similar procedure. The first report of my operative experiences was presented in 1945.[5]

Anterior spinal fusion, i.e., fusion of the vertebral bodies, has long been considered the best method of stabilizing the spine because of its mechanical efficiency. In an effort to overcome the unsatisfactory results of posterior fusion, orthopedic surgeons (Speed,[26] Mercer[18]) have in the past made attempts to devise a procedure whereby the bodies of the involved vertebrae, that is, the major weight-bearing part of the spine, could be fused. Capener,[4] in a study based on 34 cases of spondylolisthesis, concluded that the ideal operation to obtain greater mechanical efficiency would be one fixing the body of the 5th lumbar vertebra to the sacrum. He pointed out, however, that the technical difficulties of such a procedure precluded its trial.

The operation of vertebral body fusion devised by the writer consists of a subtotal removal of the intervertebral disc, including the cartilaginous plates, through a partial bilateral laminectomy. The cortical surfaces of the vertebral bodies are removed with a chisel and three or more large full-thickness bone grafts obtained from the iliac crest are driven forcibly into the interspace.

A series of 262 patients followed from 6 months to 8 years postoperatively

was reviewed in 1951 in preparation for a scientific exhibit at a meeting of the American Medical Association.[8] A complete statistical review of these cases will be the subject of another communication. Although the material has been presented on several occasions,[5–7] it has not been published. It seemed desirable to follow these cases over a long period of time and to accumulate a large enough series to be certain of my contention that this procedure is logical, practical, and has many advantages over other forms of treatment of ruptured lumbar intervertebral disc.

The number of complete and permanent cures obtained by the vertebral body fusion operation are 30 to 50 per cent higher than the "cures" after simple removal of the disc. This is attributed to the fact that vertebral body fusion eliminates the complaint of low-back pain which is the most frequent disabling postoperative symptom following simple disc removal.

INDICATIONS

The indications for spinal fusion in cases of ruptured lumbar intervertebral discs have been the subject of numerous articles in the medical literature, most of which have been written in an effort to determine which patient should be subjected to a spinal fusion and which one would get along on just a simple removal of the herniated portion of the disc. Even with our present diagnostic methods, no one has, as yet, been able to lay down specific criteria. It is true that many patients apparently remain asymptomatic for a long time following a simple disc removal. But others with exactly the same clinical picture may continue to have incapacitating low-back pain and/or develop a recurrence of the sciatica within a short time after the operation. Because of this difficulty in determining which operation a given individual may require, I have made my treatment uniform for every patient. Based on the thesis, "once a ruptured disc, always a ruptured disc," a vertebral body fusion has been done in practically every case of ruptured lumbar disc operated upon since 1944.

OPERATIVE TECHNIQUE

In the last 3 years I have received numerous requests from surgeons asking for a detailed description of the operative procedure. It is the purpose of this communication, therefore, in addition to giving the rationale of the procedure, to describe the operation as it is being done at the present time and to outline the postoperative care. The technique of vertebral body fusion from the posterior approach is not difficult for those familiar with the intervertebral disc operation and accustomed to working with the delicate structures of the nervous system in a small deep hole. By an experienced operator, the procedure can be carried out in 2 to 3 hours. At the present time, the writer performs the operation in 2 hours or less.

The procedure is actually three operations in one: removal of the bone grafts, laminectomy and removal of the intervertebral disc and lastly, the spinal fusion.

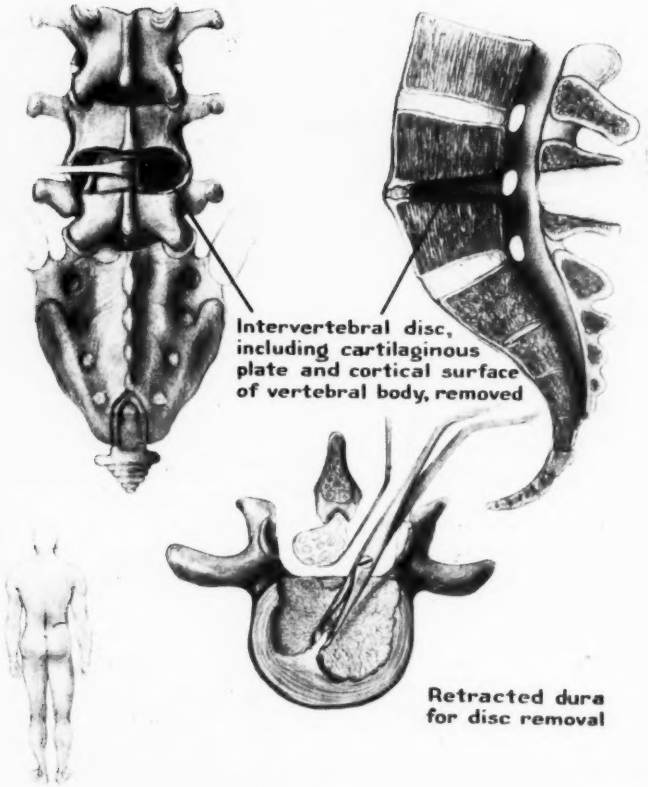
Intervertebral disc, including cartilaginous plate and cortical surface of vertebral body, removed
Retracted dura for disc removal

FIG. 1. Removal of ruptured disc in preparation for bone grafting of intervertebral space.

The bone grafts used for the fusion are obtained from the ilium, this being the ideal bone for this type of fusion. Before 1946, the bone grafts were removed from the patient's own ilium. In the latter part of 1946, experiments with banked bone were begun and since 1947, cadaver bone, preserved by refrigeration, has been used for the operation whenever it was available. The use of banked bone shortens the procedure considerably and it is appreciated by the patient because he has only one wound to heal. The technique of banked bone has been reported elsewhere.[10] To date, (September, 1952) 131 patients have been operated upon using banked bone.

The anesthetic used is intravenous pentothal sodium and 1 per cent novocain infiltrated into the skin and lumbar muscles. The patient is placed in the prone position with the spine sharply flexed and the knees bent at right angles, so that he is virtually kneeling on the table. Care must be

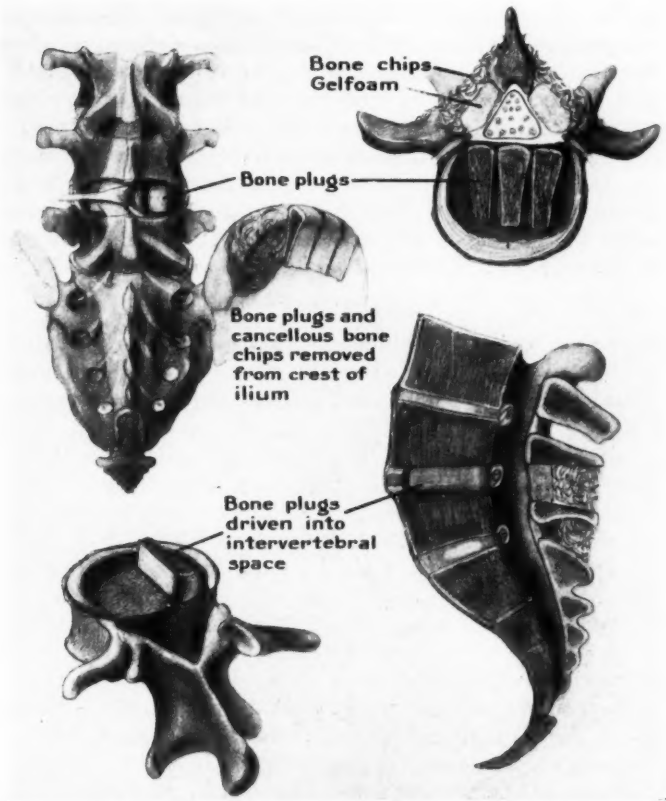
Bone chips
Gelfoam
Bone plugs
Bone plugs and cancellous bone chips removed from crest of ilium
Bone plugs driven into intervertebral space

FIG. 2. Technique of vertebral body fusion. Note wide exposure of spinal canal by removal of half of articular facets. Upper right drawing shows only gelfoam covering dura mater. The ligamentum flavum is preserved to protect dura mater.

taken not to permit pressure upon the abdomen. This causes venous engorgement, stasis and increased bleeding from the epidural veins. The patient is placed upon a sponge mattress, cut to fit the operating table. The mattress is 4 inches in thickness, with a large hole in the center. The patient's abdomen is placed over this hole. The patient is routinely given 1, sometimes 2 pints, of whole blood during the procedure.

I. THE BONE GRAFTS

A 10-cm. curved incision is made parallel to the crest of the ilium and lateral to the posterior superior spine. The sacral attachments of the gluteal muscles are cut along the crest. Care must be taken not to injure the cluneal nerves which cross the iliac crest at this point. Patients have complained bitterly of postoperative pain in the iliac incision when these

nerves have been inadvertently divided or traumatized. The muscles on the anterior and posterior surfaces of the ilium are then stripped subperiosteally with a sharp osteotome. Two bone plugs, the full thickness of the ilium, are removed. These measure 1½ by 3 cm. and are cut out at right angles to the iliac crest. If the patient has a thick ilium, another plug may be obtained, removing the cancellous bone and the posterior cortical surface, and leaving the inner cortical surface of the ilium intact. A quantity of bone chips are removed medially with the gouge and curette. Bone wax is used to arrest the bleeding from the bone surface and the soft tissues are closed with chromic gut and black silk (Fig. 2).

II. THE LAMINECTOMY

The disc operation is done through an incision and exposure for laminectomy. The spinous processes and laminae of the vertebra above and below the level of the pathology are exposed and cleaned of all soft tissue. If the pathological disc is between L4 and 5, L3, 4 and 5 and Sl are exposed bilaterally and the edges of the adjacent laminae and spinous processes of L4 and 5 are removed. Only a deep notch is made in these structures; no complete laminectomy is performed. An excellent exposure of the intervertebral space can be obtained with a minimal amount of bone removal by the use of a special instrument devised for this purpose. This instrument, called the vertebra spreader[11] (Fig. 3b and c), is a self-retaining retractor inserted between the spinous processes in the midline. The interspace can often be separated nearly twice its normal width and held in position throughout the procedure. With the vertebra spreader in place, the interlaminar ligaments are put on a stretch, and the attachments of the ligamentum flavum at the laminae and facets can be easily separated by touching them with a scalpel. An effort is made to preserve the entire ligamentum flavum, leaving it attached to the spinous process in the midline but cutting it free from the laminae above and below and from the articular facet laterally. The lateral half of the spinal canal is exposed by retracting the ligamentum flavum toward the midline like a hinge. It is held in this position by a heavy silk or catgut suture to which a hemostat is applied. This is repeated on both sides of the midline.

The interlaminar bony opening into the spinal canal is an important step in the procedure. A deep notch is made in the adjacent laminae, using a rongeur and a 5 mm. chisel. The exposure is carried as far lateral as the pedicle of the vertebra, removing half or more than half of the two articular facets (Fig. 1). The opening into the spinal canal is thus made as wide as possible. A greater exposure of the intervertebral disc is obtained and much less retraction upon the dural sac and nerve root is required for removal of the disc.

A second very useful instrument devised for this operation, the self-retaining dura retractor[9] (Fig. 3a), is then inserted. This instrument, which can be attached to any laminectomy retractor, consists of a spatula which

is engaged beneath the nerve root and dura. These structures are retracted medially to the desired position and are held in place by the turn of a screw. This permits the surgeon to use both hands to perform the operation anterior to the spinal canal. This instrument eliminates the manual retracting, pulling and hauling on the nerve root and dura and protects these structures from any injury that may possibly occur during removal of the disc and insertion of the bone grafts.

When the dura mater is first exposed by retracting the ligamentum flavum, the epidural fat is gently separated from the dura mater near the midline. The self-retaining spatula is then inserted in this line of cleavage

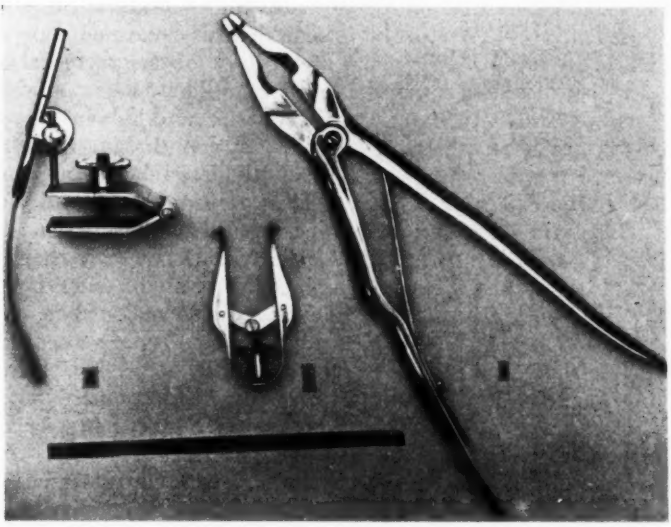

FIG. 3. (a) Self-retaining dura retractor. The clamp attaches to any laminectomy retractor. (b) The vertebra retractor and (c) vertebra spreader.

and the dura retracted medially. The epidural fat containing all of the large epidural veins then lies laterally in the spinal canal, separated from the dura and nerve root. Careful methodical cauterization of these vessels and removal of the epidural fat can be carried out with very little, if any, hemorrhage and the entire anterior wall of the spinal canal over the intervertebral disc is thus exposed, clean and dry.

A long-handled scalpel with a No. 11 pointed blade is used to remove a square plug from the posterior half of the annulus fibrosus of the disc. The knife blade is inserted to its maximum depth at the edge of the vertebral bodies and cuts are made transversely above and below the disc, medially under the dural sac, and laterally, as far as the exposure will permit, usually at or beyond the pedicle. With a strong end-biting (pituitary) disc rongeur a large plug of the disc, measuring 1×1×2 cm., is withdrawn in one piece.

This opening into the intervertebral space permits the removal of the soft parts of the disc under direct vision. A long slightly curved chisel, 7 mm. in width, is inserted beneath the cartilaginous plate and this structure is stripped easily from the vertebral body on either side of the interspace. With a sharp angulated curette and the disc rongeur, almost the entire disc is removed, leaving the adjacent surfaces of the vertebra completely clean of all soft tissue. This part of the procedure is bloodless.

It is advisable to remove with a chisel 3 to 5 mm. of bone from the superior edge of the lower vertebra. There is always an overhanging lip on the posterosuperior margin of the vertebral body. Removal of this ledge of bone makes it possible to visualize the entire superior surface of the vertebral body which may otherwise be obscured. This bone removal, which is also carried laterally to include part of the base of the vertebral pedicle, increases the width of the intervertebral space. This makes possible complete removal of all soft tissue under direct vision and aids in the insertion of the bone wedges. If marginal osteophytes are found along the rim of the upper vertebral body projecting into the spinal canal, these likewise are removed with the chisel, making sure their removal, bilaterally, extends well out into the intervertebral foramina laterally and beneath the dural sac medially. The cortical surfaces of the vertebral bodies are then chiseled off and the chisel is driven back into cancellous bone in numerous places. This assures a large, rough surface of raw bleeding cancellous bone in preparation for the bone grafts.

The dura retractor may then be removed and re-inserted on the opposite side of the spinal canal and a similar procedure carried out, removing the other half of the intervertebral disc. Or, the bone grafts may be inserted into the interspace on one side of the disc before the other half is removed. As much of the disc as possible is taken out, extending the removal to as near the anterior longitudinal ligament as possible. There is no danger of perforating the latter structures. With the vertebra spreader, one is able to see exactly how far anteriorly the disc removal is being extended. Also by forcing the spinous processes as wide apart as possible with the spreader, the anterior edges of the vertebral bodies are brought closer together.

III. THE FUSION

The bone plugs are shaped with the rongeur so that one end is slightly tapered. The pointed end is inserted into the opening in the intervertebral space, the iliac crest portion of the graft being posterior and the cancellous surface of the graft in contact with the raw surfaces of the vertebral bodies. The bone plug is then driven into the interspace with a large punch and a hammer. The self-retaining dura retractor acts as a shoehorn to protect the dural sac and the nerve root. The plug is fashioned so that, with the vertebra spreader in place, it is slightly larger in width than the intervertebral opening. By using force to drive these slightly larger plugs into position, the vertebrae are thus spread farther apart. The plug is driven about

¼ to ½ cm. beyond the posterior edge of the vertebral body, a notch having been made previously in this location when the cortical surfaces of the vertebra were removed. This small notch in the vertebral bodies and the countersinking of the plug obviates the danger of the grafts herniating posteriorly.

As many bone wedges as possible are driven into the interspace. After the first one is inserted, the interspace is widened a little more with the handle of the vertebra spreader (Fig. 3c) and, with a wide (1 cm.) chisel inserted into the interspace lateral to the first plug, the latter is gradually worked toward the midline by twisting the chisel, using the pedicle as a fulcrum, until the graft comes to lie beneath the dural sac. An opening is thus made lateral to the first graft for the insertion of a second graft or even a third if the space will permit. The same procedure is carried out on the opposite side of the dural sac so that three or four plugs or more are inserted (Fig. 4). Oozing of blood from the raw cancellous bone of the vertebral bodies after removing the cortical surfaces may be encountered. After the bone plugs have been driven into place and the vertebra spreader has been removed, this bleeding is usually completely arrested. The danger of a latent complication of bony proliferation into the spinal canal from the bone grafts is not a problem since the hard cortical surface of the crest of the ilium is in contact with the dural sac forming the anterior wall of the spinal canal.

FIG. 4. X-ray taken 10 days postoperative showing position of 3 full-thickness iliac grafts in intervertebral space, replacing the disc. The silver clip is placed to determine, by x-ray, the anterior extent of the disc removal.

Gelfoam is placed over the nerve roots laterally and the ligamentum flavum is returned to its original position, completely covering the dural sac and nerve roots. The remaining half of the two adjacent laminae and facets are chiseled up with a small gouge and fine bone chips or crumbs are placed over the interlaminar opening, thus effecting a bony closure of the spinal canal posteriorly. The wound is closed using No. 0 chromic catgut and silk.

A single rubber tissue drain is left in the wound to run off any blood or serum that may accumulate in the first 24 hours. It is then removed. A drain has been used in every case. This is contrary to the practices of many surgeons who advocate closing a spinal fusion tightly without drains. We

have observed that a large quantity of blood and serum will often escape via the drain from the wound after a spinal fusion. Patients have soaked their sheets from knee to neck. It has been my belief that this draniage from raw bleeding bony surfaces, if not permitted to escape, would accumulate in the wound and diffuse through the lumbar muscles. This could be another contributing factor to the pain and stiffness of the back so frequently noted after a posterior spinal fusion.

POSTOPERATIVE CARE AND RESULTS

The patient is placed in bed supine on two mattresses with boards on the bed springs. The writer uses a sponge rubber mattress on top of the regular mattress. Some patients may have difficulty in voiding after operation. This probably results from retraction of the sacral nerve roots during the operation or possibly may be because of the patient's position in bed (flat on his back). If he is unable to urinate voluntarily within 8 hours after surgery, a Foley catheter is inserted. After the 2nd day, he is permitted to turn himself from side to side as he desires. On the 4th postoperative day, the patient is given a mild cathartic and an enema, and the catheter is removed. Sutures are removed from the wound on the 6th day. The patient is permitted out of bed within a week after the operation and is usually discharged from the hospital on or about the 10th postoperative day.

The patients operated upon by this procedure during the first 5 years were fitted with a low-lumbar back brace which was worn for 3 months. X-rays of the spine at the end of this time usually showed a solid bridge of bone between the bodies of the vertebrae. The brace was then discontinued and the patient was encouraged to exercise his back muscles. In the past 2 years, with more extensive grafting of the interspace, we have found it unnecessary to require the patient to wear a back brace. At operation, if the stability of the intervertebral joint is tested after the bone plugs have been driven into place, the joint will be found almost completely immovable. When the patient assumes the upright position and bears weight on the joint, the grafts are locked even tighter together, thus effecting a mechanical fixation of the intervertebral joint from the beginning. Since the back brace has been discontinued, patients seem to recover the normal flexibility of their spine much sooner. Being permitted to use their back muscles from the outset, there is no danger of stiffness of these muscles from shortening due to inactivity. Early ambulation has made no difference in the rate of fusion of the vertebral bodies; if anything the fusion takes place faster. If the patient's occupation requires him to do heavy work including bending and lifting, he is permitted to return to this type of work in approximately 3 months, depending on the appearance of the fusion as determined by x-rays (Fig. 5). Many patients return to heavy work before 3 months. The patient who is not required to do heavy manual labor is often able to return to his job within 2 weeks after the operation. In this day of high cost of hospital care, the short period of hospitalization

as well as the short period of convalescence before returning to work is extremely important to the patient, economically.

The most gratifying group of patients treated by this operation has been that of the industrial accident and insurance cases. It is the experience of all surgeons who do disc surgery in industrial cases to expect long periods of postoperative convalescence with the patient drawing compensation.[22] If and when the case is closed, there is usually a fairly large settlement for

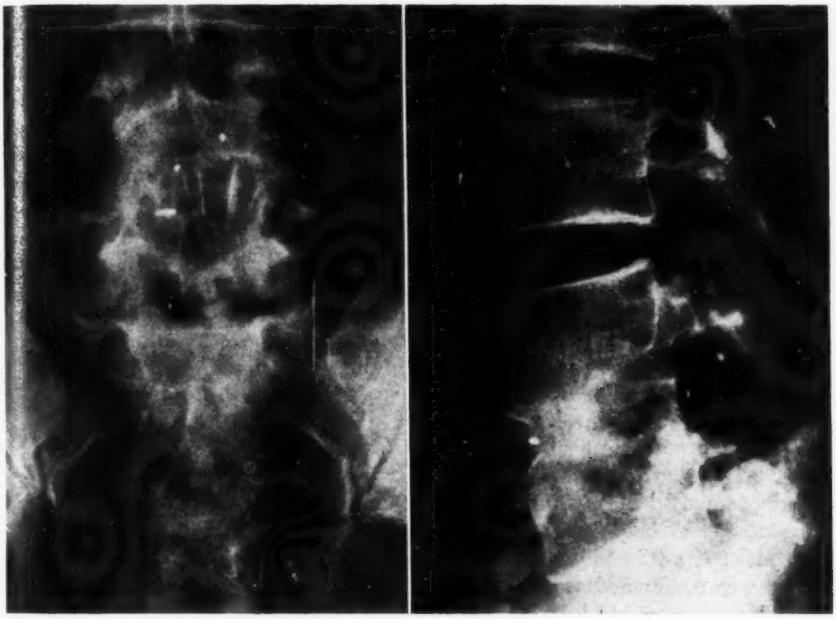

FIG. 5. X-rays taken 3½ months after surgery. Solid fusion of adjacent vertebral bodies is indicated by lines of trabeculation extending through the intervertebral graft. This patient, a "jack hammer" operator, returned to his job 2½ months after operation.

permanent partial disability.[29] My experience with the vertebral body fusion in compensation cases has been much more gratifying. In a series of 60 compensation cases, the average length of disability, that is, working days lost, was 169 days (5½ months) from the initial lay-off until the patient had recovered from the operation and returned to work. This included the early cases when the operation was not perfected as it is today. In only 5 patients was it necessary to award a permanent disability. A complete discussion of this group will be presented in another communication.

With fusion of only one vertebral joint there is little if any limitation of movement of the lumbar spine and no pain. The patient is often aware of the loss of his vertebral pain the first time he assumes the vertical position after the operation. This "vertebral" pain, which the patient will

describe as a feeling of insecurity in the lower lumbar spine, is apparently caused by loss of the support (nucleus pulposus) between two vertebrae. When this support (bone grafts) is re-established the pain immediately disappears, even before a solid fusion, i.e. bridging of the intervertebral graft, takes place.

The fusion between the vertebral bodies takes place rapidly and becomes solid in a very short time. Within 2 to 3 months the grafts can be shown by x-ray to have increased in density. This rapid fusion is aided by the fact that the grafts are placed at right angles to the vertical axis of the spine rather than parallel to it as is done in the Hibbs type of fusion. The possibility of a pseudo-arthrosis developing with the vertebral body fusion is unlikely because the fusion takes place along the weight-bearing axis of the spine. This line (through the nucleus pulposus) is the fulcrum of movement of one vertebra articulating upon another. With forward, backward and lateral action of the spine, movement is minimal at this point, and the farther from this weight-bearing center the greater the separation of the intervertebral spaces. It seems logical to assume that fusion at this point of least movement has much less chance of failure than one done at a distance from it, namely, the spinous processes and laminae. My experience with vertebral body fusion verifies these assumptions. A failure of fusion, demonstrable by x-rays, has occurred in only 6 patients.

COMPLICATIONS

In the 6 cases in which there was failure of fusion, absorption of the intervertebral grafts had occurred (unexplained), so they could not be recognized by x-ray 3 to 4 months after surgery. In 2 patients after prolonged convalescence, the vertebral bodies settled down and eventually fused. The other 4 patients continued to complain of back pain until a second fusion was performed. The experience of Van Wagenen of having the bone plugs work loose and herniate into the spinal canal has occurred in only 1 case in my series. This patient had severe sciatica postoperatively and a foot drop developed, which cleared up when the cause of the trouble was demonstrated by x-ray and adjusted by reoperation. This complication can be prevented if all cartilage and cortex are removed from the vertebral bodies before the grafts are inserted. The graft will conceivably absorb, decrease in size and work loose with movement of the spine if cartilage prevents the graft from obtaining blood supply from the vertebral body.

It has been asked if the bone plugs ever herniate or telescope into the cancellous bone of the vertebral bodies. One might expect this to occur with the hard cortical surfaces of the iliac graft in contact with what appears to be soft cancellous bone in a major weight-bearing joint. The cancellous bone of the lower lumbar vertebra, although appearing spongy, is apparently as resistant and hard as the cortex of the graft, for this complication has not occurred in my series. On the other hand, when a graft is used that is composed only of cancellous bone, without the cortical surface of the iliac

grafts, the graft is compressed with weight bearing and although an intervertebral fusion may occur, the normal width of the intervertebral space and foramen cannot be maintained as with the full-thickness iliac graft. This is of importance to assure complete success of the procedure.

Other complications have been encountered. Numbness of the foot and ankle with weakness of anterior tibial muscle have been occasionally noted. These symptoms are transient and usually disappear in days or weeks. One patient had a complete foot drop from her first operation (done elsewhere). After a second operation with fusion, the paralysis continued for 22 months, then recovered completely. These symptoms are, therefore, not considered serious. In 2 patients (early cases) there developed evidence of arachnoiditis of the cauda equina with intractable leg pain. This was attributed to lipiodol sensitivity. Bilateral upper thoracic cordotomy had to be resorted to for relief. Wound infections have been no higher than one would expect, considering the length of the operation and the use of foreign material (bone) in the wound. Infection is minimized by routine use of antibiotic drugs postoperatively. Of the 14 cases in which infection occurred it was necessary to remove the bone grafts in only 2. In all the others the wounds healed without loss of bone and went on to a more solid fusion than those that were not infected.

SUMMARY AND CONCLUSIONS

A detailed description of the operative technique for fusion of the vertebral bodies after removal of a ruptured lumbar intervertebral disc is presented. The rationale of such a procedure based on the anatomical physiology of the disc is discussed. The author's experience with this operation in a large series of cases (321 to the present time) followed over a period of 8 years is described, including improvements in technique. The after care as well as complications that have been encountered are discussed. The rapidity with which patients recover from this operation and the high percentage of complete long-term cures (over 85 per cent) seem to justify the conclusion that this operation is the treatment of choice in the care of the patient with a ruptured lumbar intervertebral disc.

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SENSITIZATION OF THE SPINAL CORD OF THE CAT TO PAIN-INDUCING STIMULI*

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SENSITIZATION of a functional unit of the central nervous system by partial injury or destruction is well known. Cannon and Haimovici[3] in 1939 reported that motoneurones could be sensitized by partial "denervation." Cannon and Rosenblueth[4] described the sensitization of both cholinergic and adrenergic elements in the autonomic system following injury to its neurones. Cannon[2] then postulated a "*law of denervation*" for the efferent systems which required sensitization as the result of partial destruction. Drake and Stavraky[5] then carried this concept into the afferent systems and showed that deafferentation caused systems to react more quickly and more intensely than did normal units. In their chronic cats, injection of either cholinergic drugs, camphor or metrazol produced convulsions which appeared earlier and more intensely in deafferented limbs than elsewhere.

Clinically, lowered threshold and increased sensitivity may result from direct sensitization of cells within a system. Injury to dorsal root ganglia is thought to cause pain in this way and trauma can so sensitize the cells of the cerebral cortex that epilepsy results from minor stimulation. Such cortical sensitization may follow destructive lesions and scar formation. It may be relieved in man by the removal of the scars.[12] Chronic experimental epilepsy, produced by the application of alumina cream to the cerebral cortex in monkeys,[10] must be the result of similar processes.

When peripheral nerves are damaged sensitization of the sensory system which mediates pain may result in hyperalgesia or causalgia. These painful states are most often unrelieved by the removal of the original source of the disorder at the site of initial injury. It has therefore been assumed that the initial injury has in some fashion sensitized the central nervous system. The concept of the neuron pool which effectively perpetuates the afferent input has been developed here.[6,11]

The presence of hyperalgesia in cats as the result of injection of alumina cream into the spinal subarachnoid space may be considered as an example of chronic sensitization of an afferent complex within the central nervous system. It has been reported in detail elsewhere.[7,8] The present paper is the result of subsequent investigations and will deal with the nature of this sensitization. A brief review of the development of the hyperalgesia as previously reported is here necessary.

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A part of this work was completed at the University of Oregon Medical School.

Nature of the Hyperalgesia. In the cats described in the previous papers hyperalgesia developed according to a fixed pattern which was similar in each animal. Following the injection of the colloidal substance into the dorsal spinal subarachnoid space, segmental skin areas became greatly sensitized to any form of cutaneous stimulus. Light touch or pin-prick, which produced only mild responses if applied to normal skin areas, caused extreme reactions of displeasure in the affected regions. Blowing on the hair, a slight touch, or even the tension caused by the animal's moving would produce withdrawal, crying-out and, sometimes, resistance. The animals became sedentary and dirty, since they could not clean the hypersensitized fur. But their non-affected regions were clean and they remained friendly and sought petting in the normal areas. A causalgia-like syndrome was thus defined.

Rate of Development. Following injection, the hypersensitivity to touch and pain first appeared, in some animals, on the 5th postoperative day. In others, symptoms were not noted until the 3rd week. The amount of substance injected was thought to have direct relation to the rate of development, although no quantitative experiments were carried out. Following the onset, the hyperalgesia usually became intensified over a period of several weeks. Once fully established it was permanent for as long as 3 years.

Extent of Hyperalgesia. The hyperirritability to sensory stimuli appeared first just below or at the level of the injection. It then spread bilaterally during a period of about 2 weeks, becoming equally intense through all levels below the site of the injection and for one or two segments above it. This occurred whether the injection was made low in the lumbar region or high in the cervical; thus a varying amount of skin area might be involved, increasing as the injections were made higher in the cord. It was thought that this must indicate a local action of the substance at the level of injection rather than action over a long distance of cord, for, in the latter instance, the diffusion should be equal in either direction from the site of injection. No actual determination of the diffusion of aluminum ions has been made, however.

METHOD

The details of the injection of alumina cream have have been given previously, as have the method of examination and the clinical status of the animals.[7,8] In the present study as in the previous ones the colloidal alumina cream has been made for us by Dr. Lenore Kopeloff according to her original method.[10] In the present study the substance was injected into the cervical, rather than lumbar region; either into the subarachnoid space (3 cats) or directly into the lateral column of the cord of one side (5 cats). Findings have been compared with those of the earlier series.

DATA

The findings in all 8 cats are consistent although different secondary procedures often altered the course of the disorder. The following brief

clinical history of one animal describes the development of postoperative hyperalgesia.

This was a mature male cat weighing 2.9 kg. at onset of the procedure and 3.7 kg. 1½ years later. It was of reasonably tame and friendly disposition and had short hair which facilitated sensory examination (Series No. 35). On Aug. 1, 1949, under nembutal anesthesia, a small drop of colloidal alumina cream was injected into the right lateral cervical cord substance at a midcervical level.

On the 2nd day the animal had eaten but appeared rather sedentary. It stood and walked without apparent difficulty but occasionally the right hind leg showed some postural distortion which might have been proprioceptive. On the 7th day no postural deficit remained and none occurred thereafter. On this date hyperalgesia to pin-prick first appeared. It was reported as moderate and extended from the region of the neck incision down. On the right side, at the level of the injection it was markedly more intense than elsewhere. By the 11th day the hyperalgesia had increased. It was then more marked in cervical and thoracic regions than "lower down." It was bilaterally equal. This status was still present on the 21st day, but by the 25th day extreme hyperirritability was noted throughout all levels below the incision. By the end of the 6th week a state had developed that persisted unchanged for the subsequent 1½ years until the animal was killed.

At the end of the 3rd month a protocol note reads as follows: "The sensory abnormality in this animal is now very marked and easily demonstrated. The fur below the cervical segments is rough and matted. About the head and neck it is smooth and clean. The animal moves little, preferring to remain crouched in the middle of its cage where it receives the least sensory stimuli. To light touch or pain stimuli there is a prompt protest as well as a reflex jerk. Hyperirritability is extreme. It extends throughout the whole body. The right foreleg is more affected than the left. It jerks to the slightest stimulus as do both hind legs, whereas the left foreleg only occasionally shows this type of response to stimuli on the left shoulder or to light touch applied to the dorsum of the foot."

One year later: "This animal shows exactly the same characteristics as have been described many times before. It tends to remain immobile with the legs folded beneath its body and the tail wound to the side. . . . The line of poorly cleaned fur is definite on both sides."

The involvement in this animal was repeatedly found to be at the cervical level and somewhat higher on the right than on the left side. In no cats, following injection into either the subarachnoid space or within the cord substance, was a distinct line of demarcation ever demonstrable between normal and hyperirritable skin regions. Rather the border appeared to fluctuate slightly both in degree of hyperirritability and in level. In this respect also, therefore, their condition resembled the causalgic states in man.

Injection of alumina cream into the lateral cervical cord substance in 4 other animals produced like syndromes and additional evidence as to the nature of the hyperirritability. In these later cats the injection was smaller and more sharply localized, as shown by subsequent histological section. In each cat the development of intense, bilateral hyperalgesia oc-

curred which included all spinal segments caudal to the injection and one or two in a rostral direction. The march of symptoms was the same in each case. All of these cervically injected animals showed a brief transient motor disability in the ipsilateral hind leg. Following this, no motor defect was observed. But there was always a marked increase in deep reflexes which caused at times a stammering, clonic type of movement during progression, especially when the animal was forced to move quickly. Skill in fine movements was unaltered.

Various attempts were made to modify the hyperalgesia and hyperreflexia in these cervical animals. This was done either by drugs or by cord section subsequent to the full development of hyperirritability. The results are here summarized. In addition to clinical observation of the behavior of the cats and their reactions to stimuli, the hyperirritability was demonstrated electromyographically in each animal. Under light dial anesthesia, needle electrodes were inserted bilaterally into various symmetrical muscle groups up and down the spinal musculature and in the extremities. A brief electrical stimulus or a mechanical pinch would then cause a characteristic muscle twitch which was recorded on a Grass 8-channel machine. Under these conditions it was consistently easy to demonstrate that the threshold for muscle twitch was lower and the response greater in the hyperirritable than in the normal regions.

As reported earlier,[8] decerebration of a cat with sensitized lumbar and sacral regions was followed by markedly greater hyperreflexia in the hyperirritable hind legs than in the previously normal forelegs. Similar results were obtained following acute cord transection in the cervical region after sensitization of the lumbar region had become chronic.

Two cats were made hyperalgesic from the high cervical regions down. Subsequent cord transections were made at lower thoracic levels and the animals were kept alive for 5 and 7 days respectively. There was no doubt but that the reflex status below the cord transection was one of unusual and extreme hyperirritability. If the cord of a normal cat is transected at this level, deep reflexes below the transection may disappear or become greatly diminished and will remain so for several days. The reflex status then shows a gradually increasing irritability until the chronic state, usually during the 2nd postoperative week, when marked hyperreflexia appears. These same changes are those that develop, although much more slowly, in spinal man. In the hyperalgesic cats, no stage of hyporeflexia appeared and the extreme hyperirritability of skin areas persisted as shown by twitching responses to slight stimuli, although of course no subjective response was possible. Knee jerks were 4 plus from the time of cord transection. These experiments demonstrated that the state of hyperirritability was not dependent on the presence of the original site of the injection.

The effects of various drugs were tried many times without any pronounced results. These experiments were carried out in collaboration with Dr. Frederic P. Haugen, Chief of the Department of Anesthesiology at the

University of Oregon Medical School, who directed the injections and the amounts of drug used. Since we were not sure of the effective dosage, the size was increased in a series of animals until profound and characteristic changes were induced. In the case of morphine, respirations diminished and the animals finally became totally unresponsive. Procaine was pushed in one instance until a convulsion resulted. In both cases the difference between normal and hyperirritable skin areas remained.

Morphine, procaine and myanesin were each given several trials, both intravenously and intraperitoneally, in unanesthetized animals. There was never any effect on hyperirritability as demonstrated by the behavior of the cat. When dial anesthesia was given the responses to painful stimuli became less and less as the anesthesia deepened. During this interval those areas that had been first and most intensely affected remained more hyperirritable than did those later affected. That is, in the cervically injected animals, deepening anesthesia reduced the area of hyperirritability to the cervical and upper thoracic areas where it had first appeared. Under light dial anesthesia the intensity of the response to painful stimuli was diminished slightly as the result of injection of morphine or procaine. Myanesin never produced any such effect.

Additional information as to the type of change produced within the nervous system was obtained from histological sections. When the substance was injected into the cord the site and extent of the area injected was verified by serial sections stained by the Nissl method. Marchi and Nissl stains of sections from above and below the level of the injection gave added information. Rostral to the lumbar subarachnoid injections there was some degeneration of the axons lying in the posterior and posterolateral columns. It was spotty and slight and was never seen as any large destruction of any tract. It is notable that it appeared in posterior columns although no lasting defect in proprioception could ever be shown. It is notable also that the degeneration in ventral and ventrolateral columns was less. Since the Marchi method stains only myelinated fibers, no information as to unmyelinated fibers is available. Weigert preparations of sections made on a number of these cats with older lesions were unproductive; the degeneration is probably too scattered and fine to show by this method. Silver staining, not yet available, might give more definite results.

DISCUSSION

Examination of the experimental hyperalgesia produced by the discrete injection of alumina cream into the cervical cord substance in the cat has confirmed the previous findings resulting from injection of larger amounts into the spinal subarachnoid space at lumbar levels. It can now be concluded that the injury is directly upon the axons of the afferent tracts transmitting pain and that this change in some way permanently sensitizes the afferent system to which these axons belong. This deduction is based on the nature of the changes, which are to be compared with those resulting from pe-

ripheral nerve injury, and upon the extent of the involvement throughout the segments caudal to the injection.

Changes confined to the very sensitive dorsal nerve roots or their ganglion cells or to the cell groups within the cord at the level of injection are not possible, as the extent of hyperalgesia would then be limited also to this level. If the nerve roots were the source of the irritation, moreover, a sharper line of demarcation between affected and non-affected areas might be expected.

The inference from physiological observations is that the fine myelinated and the non-myelinated fibers are those that are altered, since it is these that are known to transmit pain in man, and that the thicker motor and proprioceptive fibers, which are more heavily myelinated, have been more resistant to the action of the aluminum. This is borne out by the histological findings in the cords, which show degeneration of scattered fine fibers rather than of compact tracts. In sections stained by the Marchi method this degeneration lies chiefly in dorsal and dorsolateral tracts which is also consistent with what is known of the afferent fibers in the cord of the cat. In this animal, bilateral ventral cordotomy has no effect on pain perception, whereas bilateral section of dorsal quadrants consistently produces hypalgesia.[9] The tracts transmitting pain therefore lie in the dorsal quadrants of the cat's cord.

In the cat, furthermore, it has been shown that transmission of pain in the cord substance is bilateral. Ranson and von Hess[13] in 1915 first demonstrated that hemisection of the cord in the normal cat had no effect on pain sensation. This observation has been confirmed by the writer[9] by hemisections performed on both normal and hyperalgesic cats. The fact that, in the present series, hyperalgesia first appeared ipsilateral to the injection and at that level, spreading later to both sides and caudally, must mean that there is some lamination or arrangement of fibers that makes those that are first affected consistently more vulnerable than those that are later involved.

The nature of the action on the axons might be one of several. Anatomically the tracts might be totally or partially damaged. The fibers might be destroyed or merely altered. All or part of the system might be involved. Analogous hyperalgesia occurs when peripheral nerves are damaged and the evidence there seems to be that pain follows when nerves are stretched or crushed rather than cleanly severed. The extensive literature on this subject has been discussed in detail by Sunderland and Kelly.[14]

In the partially destroyed system the remaining axons may function in one of several ways. At the level of the injury, axons may be producing a constant stimulus which results in overreaction when there are additional stimuli from the environment. Or the hyperalgesia may be caused by a lowered threshold at the site of injury. The state of hyperirritability herein described, however, can not be the result of either of these possibilities alone since hyperreflexia remained in the sacral segments of a cord previously sensitized by a cervical injection, after that area of injury had been discon-

nected by complete cord transection at an intervening thoracic level. Potentiation of the entire segmental nervous system innervating the pain tracts of the affected areas must therefore have occurred.

There is plenty of evidence that such potentiation may occur. Injury to axons always produces retrograde changes in the original cell bodies which are always more marked histologically the nearer the injury is to the cell of origin. Sunderland and Kelly[14] have shown, in studying a series of peripheral nerve injuries in man, that "the higher on the pathway the level of the lesion is the more likely causalgia is to ensue." They state that "The clinical evidence suggests that the painful sequelae of nerve injuries are the effect of damage to the central nervous system by retrograde and transneural changes. A center of abnormal spontaneous activity is thereby set up in the cord which acts as a focal point for the dissemination of spreading disturbances involving wider and wider areas of the cord." There are no data at the present time that interfere with the application of this concept to the hyperirritability which is here under discussion.

The concepts of Wolff[6] and of Livingston,[11] which provide potentiation within a reverberating circuit or a neuron pool to explain referred pain and causalgia, are in accord with the present data also, although the present data require potentiation in the cord alone and not at higher levels. No evidence is available, however, to prove that potentiation may not also exist elsewhere.

Another phase of the hyperirritability deserves comment, namely that a painful state has been induced by alteration in the spinal cord. Clinically, in man, pain as the result of cord lesions is unknown although dorsal roots and ganglia are excessively sensitive to injury, from which pain always results. However, Sweet and his associates[15] have reported that pain can be experienced by unanesthetized man as the result of stimulation of the exposed cord. They produced it from stimulation of both dorsal and ventral quadrants, although its character differed in the two places. The threshold for pain from stimulation of the cord was, however, many times higher than that for stimulation of dorsal nerve roots and the character of the pain from cord stimulation was much more bearable than that from stimulation of the dorsal roots.

It should be noted here that, as far as could be observed in animals, the pain that appeared as the result of the present experiments was very like the causalgia of man. It was of marked intensity; its segmental limitations were indefinite; it could be produced by very slight stimuli which ordinarily were those felt as touch rather than pain; and it did not influence the character or psychological behavior of the subjects except when present. These animals remained peaceful and friendly, as they had been previous to operation, except when receiving disagreeable stimuli.

Analogies may be drawn between the hyperalgesic state and the experimental epilepsy which can also be produced by alumina cream.[10] It has been reported[1] that the histological change produced in the cortical tissue by

alumina cream is "essentially that of a chronic progressive meningocortical cicatrix," but that a similar type of lesion was produced by control preparations, using substances other than aluminum, which failed to induce convulsive seizure. Thus, no histological change that is pathognomonic for the aluminum substance, which is the only substance producing this irritation, has yet been found. But, if the changes in the cerebral cortex are the result of action upon the small myelinated or non-myelinated fibers, as they appear to be in the cord, and if the other substances can be shown to produce no such changes, the difference in resulting symptomatology may be explained. Since changes in small fibers are most difficult to detect it is very possible that their specific degeneration in the cortex in relation to epilepsy has been missed. The concept of a fine-fiber meshwork which may be altered by injury within the cortex is entirely possible in relation to what is known about the clinical manifestations of epilepsy. The potentiation of its cells of origin may underlie its abnormal activity.

In both its cortical and cord effects colloidal alumina cream might then be unique in producing by chemical means a partial destruction of those anatomical systems composed of fine and little-myelinated fibers and a resultant permanent hyperirritability. If this is so, then the substance should also be effective on the autonomic systems and in the peripheral nerves and in both places should produce afferent hyperirritability which might resemble clinical states in man.

CONCLUSIONS

1. The production of permanent hyperalgesia following injection of alumina cream at the cervical levels of the cord in the cat has been established.

2. This hyperalgesia involves all areas caudal to the level of injection and one to two segments rostrally.

3. There is no distinct level between normal and hyperalgesic skin and there is some fluctuation in degree of hyperalgesia. In both these attributes and in its severity this hyperalgesia in cats resembles causalgia in man.

4. The hyperalgesia cannot be attributed to potentiation of parts of the central nervous system lying rostral to the lesion since it persists following decerebration and following section of the cord both above and below the level of injection.

5. The hyperalgesia must therefore be caused by potentiation of neurones remaining active within the partially destroyed segmental system of the cord. In this respect it resembles the conditions in man which may follow injuries to peripheral nerves.

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CASE REPORTS

CAROTID CAVERNOUS FISTULA WITH SIGNS ON CONTRALATERAL SIDE

CASE REPORT*

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(Received for publication July 21, 1952)

The fact that an arteriovenous fistula between the internal carotid artery and the cavernous sinus, because of anatomical variations, is apt to present clinical findings on the side opposite to that of the pathological lesion, has been previously reported by Doctors Dandy and Walsh. However, their cases were not verified by arteriography and, to the best of our knowledge, there is only one other case (personal communication of Sir Geoffrey Jefferson from England) in which careful and well done arteriographic studies demonstrated evidence of an internal carotid-cavernous sinus fistula on the side opposite to that on which the clinical signs were present.

CASE REPORT

#064436. S.E., a 54-year-old right-handed white woman, was admitted to the Neurosurgical Service of the Neurological Institute on Oct. 21, 1951. Her chief complaint was a pounding noise inside her head, related mostly to the left ear, since May 31, 1950.

On May 30, 1950, approximately at midnight, and for an unknown reason, she had fallen while going up the front stairs of her house. She was unconscious for 2–5 minutes and there was no apparent injury resulting from the fall. The patient went to sleep and the next day as usual she was at work. During the whole day she complained of headache and pain over her eyes, and the left eye began to feel "swollen." She returned home and then about 10:00 P.M. on May 31, 1950 she noticed the sudden onset of a pounding noise "like hammering" inside her head, mostly localized to her left ear. This was a continuous noise, with exacerbations synchronous with her pulse. Immediately afterward her left eye became "harder" and completely closed, remaining that way for about 5 days. At the same time there was marked ecchymosis around the left eye, which progressively subsided in a few days. The left-sided ptosis gradually improved during the next 4 months. When her left eye began to open she noticed "double vision." This disappeared within a period of 2 months. Since May 31, 1950 her left eye had appeared to be "blood shot," mostly in the outer portion of the eyeball. There had been progressive protrusion of the left eye since the onset of her illness.

Examination. The left upper eyelid was full with congested veins. The conjunctiva showed generalized chemosis, greater in the left lower quadrant. There was a left, nonpulsating exophthalmos of 2 mm. Auscultation over the closed left eye revealed a loud bruit synchronous with her pulse. The bruit was not audible over the right eye. The bruit was not eliminated by left, right or bilateral carotid compression. Visual acuity: O.D. 20/20; O.S. 20/70 (not corrected). Visual fields were normal. Ophthalmoscopic examination showed engorgement of the veins of the left fundus. There was no papilledema. The III, IV and V cranial nerves were normal. There was no diplopia, but there was weakness of the left external rectus muscle. The rest of the cranial nerves were normal.

Laboratory Studies. Complete blood count, urinalysis, sedimentation rate, and Mazzini

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test were negative. The CSF pressure, cytology and chemistry were normal. Calorics and audiometrics were essentially normal. Skull x-rays, including special projections, were reported as negative.

Impression. Considering the history and physical findings, the diagnosis of arteriovenous fistula of the left internal carotid artery and the left cavernous sinus was made by all observers.

Arteriography. On Oct. 23, 1951, under local anesthesia, left common carotid percutaneous arteriography was performed. This failed to show any abnormality. The right common

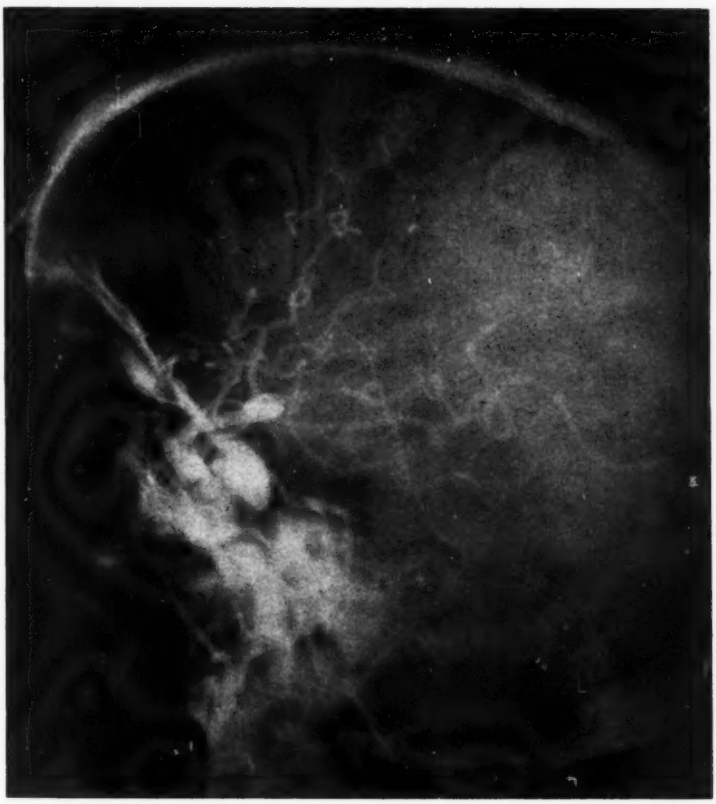


FIG. 1. The right carotid arteriogram shows the arteriovenous fistula between the right internal carotid artery and the cavernous sinus.

carotid artery was then entered and films revealed a large arteriovenous fistula in the right cavernous sinus. This drained through the anterior portion of the circular sinus into the opposite cavernous sinus and left superior ophthalmic vein (Figs. 1 and 2). Since the bruit was not obliterated by bilateral common carotid compression, percutaneous right vertebral arteriography was done. This showed a tremendous right posterior communicating artery supplying part of the carotid circulation, and explained why the bruit could not be eliminated by bilateral carotid compression (Fig. 3).

FIG. 2. The basilar view of the right carotid arteriogram shows the arteriovenous fistula on the right draining through the anterior intercavernous sinus to the left cavernous sinus, and thence forward through the greatly enlarged superior ophthalmic vein on the left side.

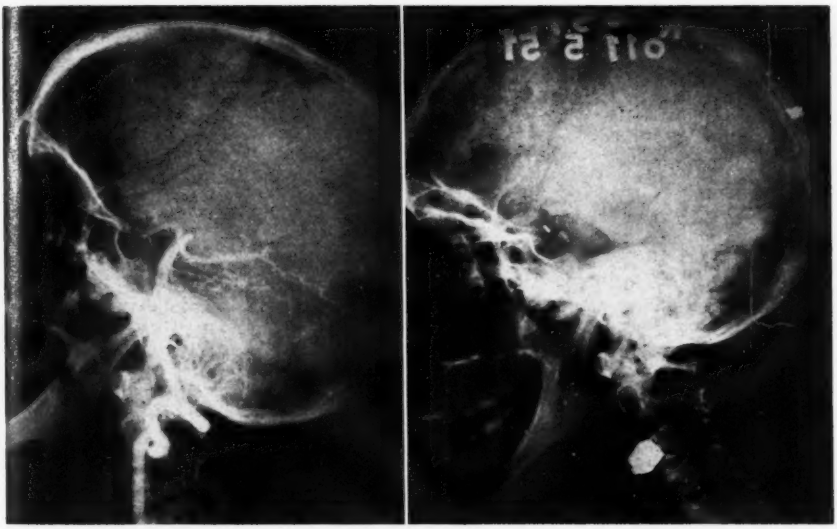
1st Operation. Two days later, on Oct. 25, 1951, the right internal carotid artery was exposed in the cervical region and a Selverstone clamp was applied. At that time a positive Babinski sign on the homolateral right side was noted before closure of the artery. During operation attempts had been made to take intra-arterial pressure recordings from both carotid arteries and for this purpose a catheter had been twice inserted in the left common carotid artery and once in the right common carotid artery. Following this, the Selverstone clamp was closed until complete obliteration of the right internal carotid artery was obtained. Since the right Babinski sign persisted and this was attributed to arterial spasm related to the two punctures of the left common carotid artery producing a reduction in the blood flow into this vessel and its branches, the Selverstone clamp was released and the wound was closed in a routine fashion. The patient was followed at 5-minute intervals and in 2 hours the right Babinski sign had disappeared. She continued to be closely observed and in view of the persisting negative neurological findings, the right internal carotid artery was completely closed with the Selverstone clamp 17 hours after the artery had been exposed.

The *postoperative course* was uneventful and one examiner was still able to hear a bruit which was very soft as compared with the pre-operative one. However, the patient still complained of the "annoying noise inside her head." The Selverstone clamp tools were re

moved 72 hours after complete obliteration of the right internal carotid artery was accomplished.

2nd Operation. On Oct. 31, 1951, through a right temporal craniectomy, the right internal carotid artery was ligated intracranially. During the operation the corresponding posterior communicating artery was well visualized (Fig. 4).

Course. The patient showed a remarkable improvement which has persisted to date. The bruit objectively disappeared although the patient described a very faint "sizzling" noise in her left ear. The conjunctival chemosis subsided as well as engorgement of the



FIGS. 3 and 4. (*Left*) The percutaneous vertebral arteriogram demonstrates the large right posterior communicating artery. The carotid arteries were not compressed when the diodrast was injected, hence none entered the cavernous sinus. (*Right*) A postoperative film shows the Selverstone clamp on the internal carotid artery in the neck, the silver clip on the internal carotid artery intracranially, and the temporal craniectomy opening through which the clip was placed on the artery.

veins of the left fundus, and the full veins previously noted in the left upper eyelid disappeared. The left proptosis clinically improved although exophthalmometer readings were the same. Visual acuity and visual fields were unchanged.

SUMMARY

1. A case is presented of a fistula of the right internal carotid artery into the right cavernous sinus, draining through the anterior part of the circular sinus to the left cavernous sinus and left superior ophthalmic vein. The clinical findings, however, suggested that the fistula was on the contralateral or left side. Apparently the patient's right ophthalmic veins did not join with the cavernous sinus.

2. The bruit over the left eye was not obliterated by bilateral carotid compression because of a large right posterior communicating artery which carried blood to the fistula from the basilar artery.

3. This case emphasizes the importance of complete arteriographic studies in

order to obtain an accurate diagnosis of the side of the lesion and of the collateral circulation, both of major importance for proper surgical therapy.

4. The use of the Selverstone clamp made this patient's management easier, considering the presence of arterial spasm on the side opposite to ligation, and prevented an additional operative procedure.

5. The surgical treatment of arteriovenous fistula between the internal carotid artery and the cavernous sinus, as described by Dandy, proved to be satisfactory in this particular case.

LEFT FRONTOPARIETAL MENINGIOMA WITH QUADRIPLEGIA

REPORT OF CASE

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Herniation of varying degrees of the tonsilla cerebelli into the upper cervical canal from increased intracranial pressure commonly occurs. A herniation of the tonsilla from a large supratentorial left frontoparietal meningioma which caused compression of the upper cervical cord and medulla and resulted in quadriplegia as an initial neurologic sign and symptom appears to be of sufficient interest to record.

It must be emphasized, however, that cases of quadriplegia or paresis have been reported, either as a result of multiple meningiomas or meningiomas in the vicinity of the foramen magnum. Ecker[1] reported a case of meningioma which arose from the clivus, anterior to the medulla, in which there was involvement of all extremities. Piehl, Reese and Steelman[2] in reporting 3 unusual cases of meningioma, described one case in which the meningioma was located at the level of the foramen magnum and resulted in the motor involvement of all extremities.

The following case is presented because of the puzzling and bizarre symptoms and signs which resulted from a single left frontoparietal meningioma that weighed 310 gm., and also to demonstrate the efforts that were necessary for the successful recovery of the patient.

A 44-year-old white woman in a semicomatose condition was admitted to the hospital on July 7, 1948. Her history was obtained from her family. Until her husband died in 1940 from an automobile accident, she had apparently been in good health. Since that time, however, she had become very nervous but was apparently well in every other way and was able to manage her own affairs.

Three years before admission the patient had remarried and had been doing well except for apparent nervousness which persisted and was noticed by her husband and relatives. In March 1948 she fell on the ice but did not lose consciousness. Since that time, however, she walked with some difficulty and had fallen several times because of weakness of the legs. This weakness became more pronounced during the last 2 months, and 2 weeks before admission the patient could not arise from a sitting position; she noticed that her arms were becoming weak and that she was unable to use her fingers or feed herself. She also had difficulty in swallowing and began to have sphincter disturbance of the rectum and bladder. Further questioning of the husband revealed that the patient had complained of a severe

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headache 6 months previously but was free from headaches when symptoms of motor power disturbance began. There was no history of convulsions.

Examination. The patient appeared well nourished but was in a semicomatose condition from which she could be aroused only by painful stimuli. Her speech was of the bulbar type. Percussion, palpation and auscultation of the skull were normal. The pupils were equal and reacted to light and accommodation, the visual fields appeared normal by gross tests and the optic disks showed a moderate papilledema with secondary atrophy. A slight emotional facial weakness on the right side and a marked nuchal rigidity were present. Sensory changes were not noted. The patient was unable to swallow water without aspirating it into the trachea. The four extremities were spastic. Spasticity was greater on the left side than on the right and was more marked in the lower extremities than in the upper. The patient was unable to move her arms or legs without support and was unable to raise her shoulders or flex her arms higher than the level of the breast on either side. The movement of her right

FIGS. 1 and 2. Ventriculograms. (*Left*) Anteroposterior. (*Right*) Posterior anterior.

hand was somewhat better than the left; however, she could not grasp objects. There was a bilateral positive Hoffmann reflex which was more marked on the left. The Babinski reflex was bilaterally positive. The deep reflexes were markedly hyperactive throughout and greater on the left side. The presence or absence of ataxia could not be determined because of the weakness of the extremities. A definite sensory level could not be outlined; however, there were vasomotor changes of the skin below the clavicle. There was a generalized atrophy of the arms and legs to a slight degree. The anal sphincter was relaxed and the reflex was absent.

Roentgenograms of the skull demonstrated increased vascularization in the left frontoparietal region. There was no evidence of a platybasia and the pineal body could not be visualized.

A presumptive diagnosis of a tumor in the region of the foramen magnum was made.

Ventriculograms demonstrated a considerable degree of displacement of the ventricular system toward the right with depression of the anterior portion of the left lateral ventricle as well as a marked displacement of the bodies of the ventricles to the right (Figs. 1 and 2).

1st Operation. A craniotomy was performed (J.L.P.) immediately after ventriculography and a large left frontoparietal meningioma was removed.

Course. The patient responded immediately after she had recovered from the effects of the anesthetic agent. She improved slightly and appeared more alert and aware of her environment but failed to recover the use of the extremities. After several days she became less responsive as her condition slowly began to deteriorate. It was believed that there was a possible hematoma in the tumor cavity or another meningioma in the region of the foramen magnum.

2nd Operation. On July 19, 1948, the left frontal craniotomy flap was re-elevated and a moderate-sized extradural hematoma was removed.

Course. The patient responded for a few hours after this procedure but within 24 hours again showed signs of loss of consciousness.

3rd Operation. On July 20, the bone flap was again elevated and then removed. At this time bleeding was not evident, the decompressed region was soft, and there was no evidence of increased intracranial pressure.

Course. She responded after this procedure but again lost consciousness on July 22.

4th Operation. The patient was taken to the operating room and as she was turned in a prone position on the operating table, respirations ceased; however, the pulse continued. Intubation was immediately performed and artificial respiration was instituted with very little preparation. A decompression of the foramen magnum and the 1st and 2nd cervical vertebrae was carried out. Upon incising the dura, which was tense, there was considerable bulging of the tonsilla through the initial opening. As soon as the dura had been opened widely over the first two segments of the cervical cord and the foramen magnum, the respirations became spontaneous and the patient resisted painful stimulation.

Course. The return of motion in the extremities was rapid, as was the rate of recovery, and on the 12th postoperative day the patient was able to walk out of the hospital unassisted.

She was last seen on April 3, 1952, at which time there were no neurologic changes except for a slight pallor of the optic disks. There was a defect in the skull as a result of the removal of the bone flap but the patient has hesitated in permitting a cranioplasty up to this time.

SUMMARY AND CONCLUSIONS

A case report of a woman who had a large left frontoparietal meningioma has been presented. The clinical course was complicated by herniation of the tonsilla cerebelli, with compression of the upper cervical cord resulting in a definite quadriplegia.

Meningiomas may produce bizarre and puzzling symptoms. A single supratentorial meningioma that produces a quadriplegia is a rare entity. In our experiences with brain tumors at the Lahey Clinic, this is the only patient in whom it has been necessary to decompress the upper cervical cord and medulla because of compression by herniated cerebellar tonsil into the upper cervical canal.

A thorough search for identical case reports in the medical literature by one of us (E.W.S.) has been unproductive.

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EOSINOPHILIC GRANULOMA OF BONE

REPORT OF A CASE

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Eosinophilic granuloma of bone was first reported as a clinical entity in 1940 by Otani and Ehrlich[4] and independently by Lichtenstein and Jaffe.[3] In 1942 Green and Farber[1] and later in 1944, Jaffe and Lichtenstein[2] advanced the idea that Hand-Schüller-Christian's disease, Letterer-Siwe's disease and eosinophilic granuloma are allied, but these authors differed on the question of etiology. The present case is reported because of the unusual size of the granuloma, the presence of multiple bone lesions and the accompanying thrombocytopenic purpura, which has been reported as a feature of Letterer-Siwe's disease in infants and children.

REPORT OF A CASE

A 57-year-old Jewish housewife was admitted to the Gallinger Municipal Hospital with a draining sinus at the vertex, of 6 months' duration. The past history revealed a hemorrhagic tendency with excessive bleeding when cut, easy bruising and frequent nosebleeds since the age of 5. One year prior to admission a painful area developed at the vertex shortly after the patient had had several teeth extracted. This finally opened spontaneously and began to drain. At the same time a draining sinus developed on the right mandible near the chin, together with tinnitus of the left ear and tenderness over the left mastoid. The lesion at the vertex was associated with intermittent pain.

Examination revealed an ulcer at the vertex, 2.5 cm. in diameter, and an area of softening under the scalp, extending over both the posterior parietals and anterior portion of the occipital bones and measuring 15 cm. in diameter. A draining sinus was also present on the right mandible and a serosanguineous discharge came from the left auditory meatus. There were numerous petechial hemorrhages over the abdomen and areas of purpura and ecchymosis over the extremities. Neurologic findings were normal.

The initial laboratory studies revealed no abnormalities except for a platelet count of 20,900. A biopsy of the margin of the scalp lesion and underlying tissue was reported as "chronic granulation tissue." Bone marrow studies showed an increased megakaryocyte count with a decreased platelet production. Bleeding time was prolonged. Roentgenograms revealed an extensive area of bone destruction in the skull with no new bone formation (Fig. 1).

Course. A splenectomy was done in an attempt to correct the thrombocytopenia. Six weeks later the patient's platelet count was still only 36,000. Nevertheless, because of the tremendous size of the bony defect, surgery was decided upon. The preoperative diagnosis was "chronic osteomyelitis" of the skull.

Operation. Under endotracheal anesthesia an incision was made from ear to ear over the vertex of the skull, similar to a postmortem incision. The scalp was stripped back, exposing a large cranial defect with ragged borders and a great deal of granulomatous tissue adherent to the dura mater. This was stripped off in sheets and there was considerable bleeding. The bone, also, was very vascular and the procedure of biting it away was accompanied by continuous blood loss. A defect which measured approximately 15×15 cm. was thus created. Because of the favorable reports on the use of tantalum plates for acute skull defects, we decided to insert a tantalum plate as a primary procedure in the hope that any further infec-

tion might be controlled by massive doses of antibiotics, and that the patient could weather through the procedure. Accordingly, a 15 by 15 cm. tantalum plate, neither trimmed nor otherwise altered except for perforating it and bending it to conform to the shape of the skull, was fitted in over the defect (which even now was not fully covered) and secured in place with tantalum screws. Beneath the plate a drain was placed and led out through a stab incision.

During the operation the patient received 3500 cc. of blood and lost at least an equal amount. However, upon leaving the operating room she was awake and responded slowly though her blood pressure was 70/40.

Pathological Examination. The report by the pathologist revealed for the first time that the lesion was an eosinophilic granuloma (Fig. 2).

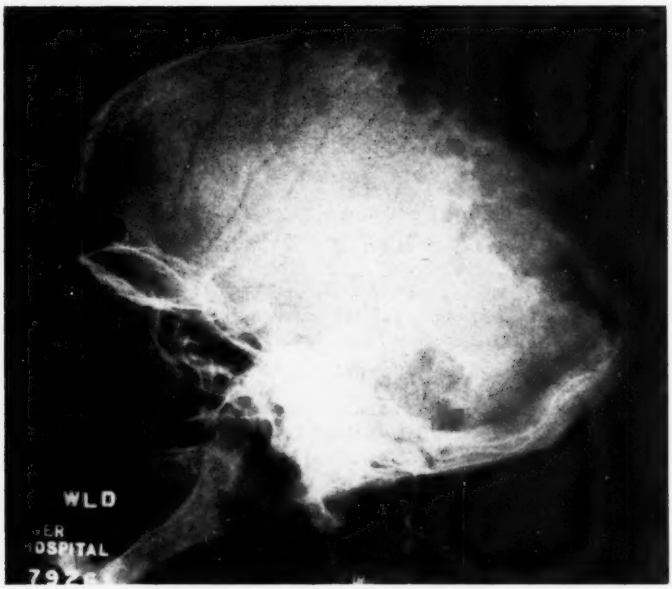

FIG. 1. Lateral roentgenogram of skull showing extensive involvement of the calvarium by eosinophilic granuloma. Note also destruction of the mastoid. (The lesion in the jaw is unfortunately covered by the film markings.)

Course. Two weeks postoperatively the original draining sinus at the vertex reopened though the initial surgical incision healed per primam. The sinus was again excised and closed 1 month later but after about 2 weeks it again broke down and drained. Shortly after the healing of the new excisions a course of deep roentgen therapy, totalling 1350 R units (450 R through three ports) was given, with a dramatic change in the X-ray picture in 1 month's time. However, shortly after the X-ray therapy was given, again the central wound broke down. In spite of this the patient insisted on being discharged with a draining sinus at the vertex and the plate plainly visible.

Three months later she was re-admitted for removal of the plate. The initial coronal incision was reopened, the scalp was stripped back and the plate was removed from its bed. Between it and the dura mater there was a small amount of granulation tissue. The coronal incision was then closed. The site of the old draining sinus was then sutured and in accom-

lishing this, the scalp had to be pulled tightly across the brain. Blood loss was minimal.

As the patient was being lifted from the face-down cerebellar position to be placed on the cart, she suddenly stopped breathing though the pulse and blood pressure remained unchanged. Artificial respiration via the endotracheal tube was immediately instituted. After about 3 minutes the heart sounds became weaker, and the patient was turned on her side and the dressing was removed, as were the sutures in the scalp defect. There was an immediate rush of a large amount of unclotted blood and the heart sounds immediately became better. Since there was no method of applying pressure, the scalp continued to bleed steadily with the blood remaining liquid and not forming any clots. In the next 5 hours 3500 cc. of blood

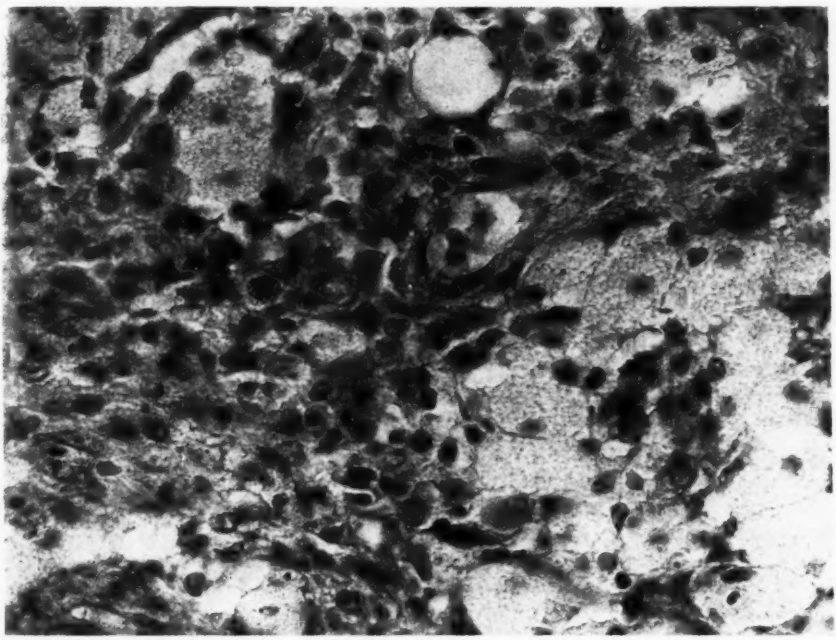

FIG. 2. Photomicrograph of tissue removed from the skull at operation. There are scattered collections of foam cells and a large number of eosinophiles and histiocytes characteristic of eosinophilic granuloma. (Hematoxylin and eosin, ×250.)

were given in the operating room, but the blood loss continued steadily and uncontrollably. The patient never regained consciousness and succumbed 8 hours after operation. She had been placed in a Drinker respirator upon her removal from the operating room. Unfortunately, autopsy was denied.

Comment. We speculate that with the absence of support to the brain over this extensive occipitoparietal area either inadvertent compression of the brain was caused by the bandages or by subgaleal hemorrhage, or that in turning the patient over on her back a sudden shifting of the cranial contents occurred with torsion of the brain stem at the incisura, producing an acute decerebration and early death.

SUMMARY

A case of eosinophilic granuloma is reported, involving multiple areas of bone and associated with thrombocytopenic purpura, such as is seen in Letterer-Siwe's disease.

The extensive cranial involvement in this case by the granulomatous process may possibly be the largest single lesion yet reported.

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1944 OLJENICK, IGNATIUS W. 435 East 57th St., New York, N. Y.
1944 OLSEN, AXEL 115 Linwood Ave., Ardmore, Pa.
1950 OSHER, DANIEL 871 Doctors Bldg., Cincinnati 2, Ohio
1945 OTENASEK, FRANK J. 6 East Eager St., Baltimore 2, Md.
1952 PACE, JOHN WILLIAM 1308 Olive Ave., Fresno 4, Calif.
1952 PAGE, W. RANDOLPH 4900 St. Charles Ave., New Orleans, La.
1952 PAGET, JOHN FRANCIS 270 Commonwealth Ave., Boston 16, Mass.
1952 PALAZZO, FRANK ANTHONY 16 Hampton Village Plaza, St. Louis 9, Mo.
1951 PARENTEAU, ANDRE 847 Cherrier St., Montreal, Canada
1952 PARKINSON, DWIGHT Medical Arts Bldg., Winnipeg, Manitoba, Canada
1949 PARSONS, HERBERT 57 East 93rd St., New York 28, N. Y.
1947 PATTON, WILLIAM B. 906 Government St., Mobile 20, Ala.
1952 PEACHER, WILLIAM G. Medical Arts Bldg., Syracuse, N. Y.
1940 *PEET, MAX M. Ann Arbor, Mich.
1943 PENFIELD, WILDER 3801 University St., Montreal 2, Canada
1951 PERLMUTTER, ERWIN 716 DuPont Bldg., Miami 32, Fla.
1948 PERRET, GEORGE Iowa State University Hospital, Iowa City, Iowa
1951 †PETERSON, CARL A. New York, N. Y.
1940 PEYTON, WILLIAM T. University of Minnesota Hospital, Minneapolis 14, Minn.
1951 PFEIL, EDGAR T. 926 East McDowell Rd., Phoenix, Ariz.
1940 ‡PILCHER, COBB Nashville, Tenn.
1951 PITTS, WILLIAM R. Doctors Bldg., Kings Drive, Charlotte 7, N. C.
1950 POLLARD, CLAUDE, JR. 411 Hermann Professional Bldg., Houston 5, Texas
1947 POLMETEER, FRANK E. 716 Professional Bldg., Long Beach 2, Calif.
1942 POOL, J. LAWRENCE 195 Fort Washington Ave., New York 32, N. Y.
1943 POPPEN, JAMES L. 605 Commonwealth Ave., Boston 15, Mass.
1951 POWELL, CHESTER B. 60 South Fourth East, Salt Lake City 2, Utah
1948 PRIBRAM, KARL H. Burlingame Research Laboratories, Institute of Living, Hartford, Conn.
1943 PUDENZ, ROBERT H. 696 East Colorado St., Pasadena 1, Calif.
1940 PUTNAM, TRACY J. 450 North Bedford Dr., Beverly Hills, Calif.
1947 QUADE, RAYMOND H. 116 South Commercial St., Neenah, Wis.
1941 RAAF, JOHN 1010 Medical-Dental Bldg., Portland 5, Ore.
1940 RAND, CARL W. 2010 Wilshire Blvd., Los Angeles 5, Calif.
1945 RANEY, AIDAN A. 1136 West 6th St., Los Angeles 17, Calif.
1940 RANEY, RUPERT B. 1136 West 6th St., Los Angeles 17, Calif.
1951 RANSOHOFF, JOSEPH 710 West 168th St., New York 32, N. Y.
1947 RASKIND, ROBERT 1079 Madison Ave., Memphis 3, Tenn.
1943 RASMUSSEN, THEODORE University of Chicago, Chicago 37, Ill.
1942 RAY, BRONSON S. 943 Lexington Ave., New York 21, N. Y.
1940 REEVES, DAVID L. 316 West Junipero St., Santa Barbara, Calif.
1951 REHFELDT, FREDERICK C. 1001 Pennsylvania Ave., Fort Worth 4, Texas
1941 REICHERT, FREDERICK LEET Stanford University Hospital, San Francisco 15, Calif.
1951 REVILLA, ANTONIO GONZALEZ P.O. Box 1392, Panama City, Republic of Panama

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* Deceased March 25, 1949.
† Deceased October 28, 1951.
‡ Deceased September 22, 1949

1951 Reyes, Victor A. (foreign certificate) 1115 San Andres St., Malate, Manila, Republic of Philippines
1952 Rifkinson, Nathan Box 1494, San Juan, Puerto Rico
1946 Risteen, Wilford A. U. S. Navy
1950 Ritchie, Wallace Parks 917 Lowry Medical Arts Bldg., St. Paul 2, Minn.
1950 Rix, Robert Alvin, Jr. 525 N.W. 11th, Oklahoma City 3, Okla.
1948 Rizzoli, Hugo V. 1150 Connecticut Ave., N.W., Washington 6, D. C.
1941 Robertson, Robert C. L. 411 Hermann Professional Bldg., Houston 5, Texas
1950 Robson, John Theodore Medical Arts Bldg., Tacoma 2, Wash.
1952 Rosenberg, Meyer 61 Eastern Parkway, Brooklyn, N. Y.
1949 Roulhac, George E. 3720 Washington Blvd., St. Louis 8, Mo.
1940 Rowe, Stuart N. 516 Medical Arts Bldg., Pittsburgh 13, Pa.
1940 Sachs, Ernest 333 Cedar St., New Haven 11, Conn.
1949 Sanchez-G., Manuel Marsella #14, Mexico, D. F.
1940 Scarff, John E. 710 West 168th St., New York 32, N. Y.
1951 Scheuerman, Walter G. 210 West State St., Trenton, N. J.
1948 Schlesinger, Benno 1125 Madison Ave., New York 28, N. Y.
1949 Schlesinger, Edward B. 710 West 168th St., New York 32, N. Y.
1950 Schneider, Richard C. University Hospital, Ann Arbor, Mich.
1941 Schnitker, Max T. 1031 Secor Hotel Bldg., Toledo 4, Ohio
1940 Schreiber, Frederic 10 Peterboro St., Detroit 1, Mich.
1952 Schultz, Arthur Lenton U. S. Navy
1949 Schultz, E. C. 525 Physicians & Surgeons Bldg., Memphis 3, Tenn.
1942 Schwartz, Henry G. Washington University School of Medicine, St. Louis 10, Mo.
1940 Scott, Michael 255 South 17th St., Philadelphia 3, Pa.
1941 Scoville, William B. 85 Jefferson St., Hartford 14, Conn.
1949 Segerberg, Ludwig H. 405 Heyburn Bldg., Louisville 2, Ky.
1952 Seletz, Emil 405 North Bedford Dr., Beverly Hills, Calif.
1950 Selverstone, Bertram New England Center Hospital, Boston 11, Mass.
1940 Semmes, R. Eustace 899 Madison Ave., Memphis 3, Tenn.
1948 Shannon, Edward W. 1314 Hanna Bldg., Cleveland 15, Ohio
1940 Shelden, C. Hunter 696 East Colorado St., Pasadena 1, Calif.
1947 Shenkin, Henry A. 255 South 17th St., Philadelphia 3, Pa.
1952 Shepard, John W. 1554 Northern Blvd., Manhasset, N. Y.
1949 Sherman, Irving J. 1026 Park Ave., Bridgeport 4, Conn.
1952 Siefert, H. Jack 400 Twenty-Ninth St., Oakland, Calif.
1950 Silbermann, Salo J. 179 Allyn St., Hartford, Conn.
1952 Silver, Maurice L. 102 Waterman St., Providence 6, R. I.
1950 Simmons, Donald R. 815 Kales Bldg., Detroit 26, Mich.
1942 Siris, Joseph H. 61-34—188th St., Flushing, L. I., N. Y.
1951 Smith, Frank P. Strong Memorial Hospital, Rochester 20, N. Y.
1951 Smith, W. Eidson Medical Arts Bldg., Knoxville, Tenn.
1945 Smolik, Edmund A. 3720 Washington Blvd., St. Louis 8, Mo.
1940 Snodgrass, Samuel R. John Sealy Hospital, Galveston, Texas
1950 Somberg, Harold M. 31 Lincoln Park, Newark 5, N. J.
1947 Speigel, I. Joshua 55 East Washington St., Chicago 2, Ill.
1949 Spence, William T. 1150 Connecticut Ave., Washington 6, D. C.
1952 Spicer, Charles 812 Pine Ave., Long Beach 13, Calif.
1950 Spitz, Eugene B. 1740 Bainbridge St., Philadelphia 46, Pa.
1940 Spurling, R. Glen 405 Heyburn Bldg., Louisville 2, Ky.
1948 Stafford, Donald E. The Neurosurgical Clinic, 902 Boren Ave., Seattle 4, Wash.
1950 Steelman, Harry F. 2314 North 32nd St., Waco, Texas
1951 Stellar, Stanley 180 East 64th St., New York 21, N. Y.
1949 Stevenson, William D. 324 Spring Garden Road, Halifax, Nova Scotia
1940 Stookey, Byron 710 West 168th St., New York 32, N. Y.
1948 Stowell, Averill Springer Clinic, 604 South Cincinnati Ave., Tulsa, Okla.
1951 Strain, Richard E. 716 DuPont Bldg., Miami 32, Fla.
1950 Strully, Kenneth J. 20 Fifth Ave., New York 11, N. Y.
1949 Stuck, Ralph M. 632 Republic Bldg., Denver 2, Colo.
1948 Suckle, Henry M. 414 Tenney Bldg., Madison 3, Wis.

1950 SUGAR, OSCAR 912 South Wood St., Chicago 12, Ill.
1950 SVIEN, HENDRIK J. 102 Second Ave., S.W., Rochester, Minn.
1945 SWANSON, HOMER S. 384 Peachtree St., N.E., Atlanta, Ga.
1951 SWEENEY, DONALD B. 2205 Highland Ave., Birmingham 5, Ala.
1946 SWEET, WILLIAM H. Massachusetts General Hospital, Boston 14, Mass.
1944 TAEFFNER, JOHN H. 3464 West School House Lane, Philadelphia 44, Pa.
1946 TAFFEL, MAX 850 Howard Ave., New Haven 11, Conn.
1952 TARKINGTON, JOSEPH 700 North Michigan Ave., Chicago 11, Ill.
1941 TARLOV, I. M. 1249 Fifth Ave., New York 28, N. Y.
1940 TEACHENOR, FRANK R. 221 Plaza Time Bldg., Kansas City 2, Mo.
1948 TEXTOR, CHARLES S., II 8 South Michigan Ave., Chicago 3, Ill.
1949 THOMPSON, RAYMOND K. The Latrobe, Charles & Read Sts., Baltimore 2, Md.
1944 THOMSON, JAMES L. 405 Wainwright Bldg., Norfolk 10, Va.
1952 TICE, WILLIAM PRESTON 317 Carlton Terrace Bldg., Roanoke, Va.
1942 TINSLEY, MILTON 25 East Washington St., Chicago 2, Ill.
1949 TITRUD, LEONARD A. 929 Medical Arts Bldg., Minneapolis 2, Minn.
1946 TROLAND, CHARLES E. 1200 East Broad St., Richmond 19, Va.
1950 TRUPP, MASON 329 East Davis Blvd., Tampa 6, Fla.
1941 TURNBULL, FRANK 913 Medical-Dental Bldg., Vancouver 1, B. C., Canada
1948 TURNER, OSCAR A. 2204 Glenwood Ave., Youngstown 11, Ohio
1946 TURNEY, M. FRANK Medical Arts Bldg., Knoxville, Tenn.
1952 TYRER, AUSTIN ROY, JR. 22 North Manassas, Memphis 5, Tenn.
1947 UIHLEIN, ALFRED 102 Second Ave., S.W., Rochester, Minn.
1944 ULMER, JACK L. Veterans Administration Hospital, Richmond 19, Va.
1940 VAN WAGENEN, WILLIAM P. 260 Crittenden Blvd., Rochester 20, N. Y.
1941 VER BRUGGHEN, ADRIEN 122 South Michigan Ave., Chicago 3, Ill.
1947 VOGEL, PHILIP J. 1801 New Jersey St., Los Angeles 33, Calif.
1941 VORIS, HAROLD C. 30 North Michigan Ave., Chicago 2, Ill.
1940 WALKER, A. EARL Johns Hopkins Hospital, Baltimore 5, Md.
1947 WALKER, EXUM 133 Doctors Bldg., Atlanta 3, Ga.
1950 WALKER, JAMES CHRISTIE Scott and White Clinic, Temple, Texas
1950 WALLMAN, LESTER J. Mary Fletcher Hospital, Burlington, Vt.
1949 WARD, ARTHUR A., JR. University of Washington School of Medicine, Seattle 5, Wash.
1944 WATSON, C. ROBERT 1014 Donaghey Bldg., Little Rock, Ark.
1941 WATTS, JAMES W. 1911 R. St., N.W., Washington 9, D. C.
1947 WEARY, WILLARD B. 3607 Gaston Ave., Dallas 1, Texas
1951 WEAVER, EDGAR N. 317 Carlton Terrace Bldg., Roanoke, Va.
1945 WEAVER, THOMAS A., JR. 521 Third National Bldg., Dayton 2, Ohio
1950 WEBB, EUGENE MCDANIEL 384 Post St., San Francisco 8, Calif.
1945 WEBSTER, JOHN E. 801 David Whitney Bldg., Detroit 26, Mich.
1941 WEGNER, WALTER R. 818 Harrison Ave., Boston 18, Mass.
1951 WEIFORD, EDWARD C. 320 West 47th St., Kansas City, Mo.
1942 WEINBERGER, LAURENCE M. 911 Second National Bldg., Akron, Ohio
1951 WEISS, EUGENE K. 724 First National Bank Bldg., Scranton 3, Pa.
1950 WELCH, WILLIAM K. Letterman Army Hospital, San Francisco, Calif.
1944 WERDEN, DELBERT H. 501 Medico-Dental Bldg., San Diego 1, Calif.
1945 WHITCOMB, BENJAMIN B. 85 Jefferson St., Hartford 14, Conn.
1942 WHITE, JAMES C. Massachusetts General Hospital, Boston 14, Mass.
1950 WHITELEY, WILLIAM H., III Jefferson Hospital, Philadelphia, Pa.
1946 WHITFIELD, ROBERT D. 38 Willett St., Albany 10, N. Y.
1948 WIGDERSON, HENRY 41-08—74th St., Jackson Heights, L. I., N. Y.
1941 WILKINS, HARRY 525 N.W. 11th, Oklahoma City 3, Okla.
1952 WILLIAMS, JOHN R. 626 Medical Arts Bldg., Grand Rapids 2, Mich.
1950 WILLIAMS, JONATHAN M. 2014 R. St., N.W., Washington 9, D. C.
1948 WILLIAMSON, WILLIAM P. Dept. of Surgery, University of Kansas Medical Center, Kansas City 12, Mo.
1947 WILSON, ARCHER A. 1031 Quarrier St., Charleston 1, W. Va.
1952 WILSON, CHARLES M. 510 Medical Arts Bldg., Dallas, Texas
1941 WINGEBACH, WILFRID 1045 Park Ave., New York 28, N. Y.
1952 WINOKUR, GERALD L. 681 Bergen Ave., Jersey City, N. J.

1948 WOLKIN, JULIUS 2010 East 102nd St., Cleveland 6, Ohio
1943 WOODHALL, BARNES Duke University Hospital, Durham, N. C.
1943 WOODS, WARD W. 2330 First Ave., San Diego 1, Calif.
1947 WOOLF, JACK I. 3607 Gaston Ave., Dallas 1, Texas
1949 WOOLSEY, ROBERT D. 16 Hampton Village Plaza, St. Louis 9, Mo.
1949 WRIGHT, STEWART A. 310 Medical Arts Bldg., Salt Lake City, Utah
1945 WYCIS, HENRY T. Temple University Hospital, Philadelphia 40, Pa.
1952 ZOLL, JOHN G. 131 Linwood Ave., Buffalo 9, N. Y.

Summary

Total certified = 459 Living = 450 Dead = 9

GEOGRAPHICAL LOCATION OF NEUROSURGEONS CERTIFIED BY THE AMERICAN BOARD OF NEUROLOGICAL SURGERY

ALABAMA (5)

BIRMINGHAM
- Galbraith, J. G.
- Graham, S. E.
- Haynes, W. G.
- Sweeney, D. B.

MOBILE
- Patton, W. B.

ARIZONA (3)

PHOENIX
- Eisenbeiss, J. A.
- Green, J. R.
- Pfeil, E. T.

ARKANSAS (1)

LITTLE ROCK
- Watson, C. R.

CALIFORNIA (47)

BEVERLY HILLS
- Andler, M. M., Jr.
- Putnam, T. J.
- Seletz, E.

FRESNO
- Pace, J. W.

LONG BEACH
- French, J. D.
- Polmeteer, F. E.
- Spicer, C.

LOS ANGELES
- Anderson, F. M.
- Brown, M. H.
- Crockett, H. G.
- Cuneo, H. M.
- Ettleson, A.
- Jones, D. W.
- Rand, C. W.
- Raney, A. A.
- Raney, R. B.
- Vogel, P. J.

NORTH HOLLYWOOD
- Dueker, H. W.

OAKLAND
- Lawrence, L. B.
- Norcross, N. C.
- Siefert, H. J.

PASADENA
- Freshwater, D. B.
- Grant, W. T.
- Pudenz, R. H.
- Shelden, C. H.

SACRAMENTO
- Black, H. A.

SAN DIEGO
- Garrity, R. W.
- Werden, D. H.
- Woods, W. W.

SAN FRANCISCO
- Adams, J. E.
- Boldrey, E. B.
- Brown, H. A.
- Cross, G. O.
- Fender, F. A.
- Fleming, H. W.
- Garol, H. W.
- Greenwood, R. C.
- Jones, O. W., Jr.
- Levin, G.
- Lusignan, F. W.
- Morrissey, E. J.
- Naffziger, H. C.
- Reichert, F. L.
- Webb, E. M.
- Welch, W. K.

SANTA BARBARA
- Reeves, D. L.

STANFORD
- Hawkins, R.

COLORADO (6)

DENVER
- Baldwin, M.
- Freed, C. G.
- Griffin, J. G.
- Lipscomb, W. R.
- McClintock, H. G.
- Stuck, R. M.

CONNECTICUT (12)

BRIDGEPORT
- Sherman, I. J.

HARTFORD
- Buckley, R. C.
- Pribram, K. H.
- Scoville, W. B.
- Silbermann, S. J.
- Whitcomb, B. B.

NEW HAVEN
- Black, S. P. W.
- Brody, B. S.
- German, W. J.
- Sachs, E.
- Taffel, M.

STAMFORD
- McNerney, J. C.

DELAWARE (1)

WILMINGTON
- Gordy, P. D.

DISTRICT OF COLUMBIA (9)

WASHINGTON
- Fulcher, O. H.
- Gallagher, J. P.
- Lambros, V. S.
- Martin, J.
- Murphy, J. P.
- Rizzoli, H. V.
- Spence, W. T.
- Watts, J. W.
- Williams, J. M.

FLORIDA (6)

JACKSONVILLE
- Lyerly, J. G.

MIAMI
- Haverfield, W. T.
- Keedy, C.
- Perlmutter, E.
- Strain, R. E.

TAMPA
- Trupp, M.

GEORGIA (7)

ATLANTA
- Dowman, C. E.
- Fincher, E. F.
- Kristoff, F. V.
- Mabon, R. F.
- Swanson, H. S.
- Walker, E.

AUGUSTA
- Manganiello, L. O. J.

ILLINOIS (18)

CHICAGO
- Amador, L. V.
- Bailey, P.
- Bucy, P. C.
- Cascino, J. P.
- Corcoran, C. E.
- Davis, L.
- Gustafson, W. A.
- Oberhill, H. R.
- Oldberg, E.
- Rasmussen, T.
- Speigel, I. J.
- Sugar, O.
- Tarkington, J.
- Textor, C. S. II
- Tinsley, M.
- Ver Brugghen, A.
- Voris, H. C.

SPRINGFIELD
- Barringer, F. S

INDIANA (5)

FORT WAYNE
Hastings, W. C.
INDIANAPOLIS
Cure, C. W.
Fausset, C. B.
Glass, R. L.
Heimburger, R. F.

IOWA (6)

DES MOINES
Abbott, W. D.
Bakody, J. T.
Decker, H. G.

IOWA CITY
Hyndman, O. R.
Meyers, R.
Perret, G.

KANSAS (1)

WICHITA
Bacon, A. H.

KENTUCKY (6)

LEXINGTON
Chenault, H.
LOUISVILLE
Grantham, E. G.
Jelsma, F.
Marshall, T. M.
Segerberg, L. H.
Spurling, R. G.

LOUISIANA (3)

NEW ORLEANS
Echols, D. H.
Karr, H. H.
Page, W. R.

MAINE (2)

PORTLAND
Maltby, G. L.
WATERVILLE
Crawford, A. S.

MARYLAND (7)

BALTIMORE
Arnold, J. G., Jr.
Bagley, C., Jr.
Chambers, J. W.
Govons, S. R.
Otenasek, F. J.
Thompson, R. K.
Walker, A. E.

MASSACHUSETTS (22)

BOSTON
Ballantine, H. T., Jr.
Bering, E. A., Jr.
Brougham, M. F.
Dorsey, J. F.
Drew, J. H.
Horrax, G.
Ingraham, F. D.
Landry, C. L.
Lowis, S.
Matson, D. D.
Michelsen, J. J.
Mixter, W. J.
Munro, D.
Paget, J. F.
Poppen, J. L.
Selverstone, B.
Sweet, W. H.
Wegner, W. R.
White, J. C.
CHELSEA
James, W. J.
SPRINGFIELD
Cheney, R. H.
WORCESTER
Carmody, J. T. B.

MICHIGAN (16)

ANN ARBOR
Bassett, R. C.
Kahn, E. A.
Schneider, R. C.
DETROIT
Gass, H. H.
Granger, G. R.
Gurdjian, E. S.
Knighton, R. S.
Nielsen, A.
Schreiber, F.
Simmons, D. R.
Webster, J. E.
GRAND RAPIDS
Lamberts, A. E.
List, C. F.
Williams, J. R.
KALAMAZOO
Light, R. U.
PONTIAC
Gates, E. M.

MINNESOTA (12)

MINNEAPOLIS
Buchstein, H. F.
French, L. A.
Peyton, W. T.
Titrud L. A.

ROCHESTER
Baker, G. S.
Craig, W. McK.
Love, J. G.
MacCarty, C. S.
Svien, H. J.
Uihlein, A.
ST. PAUL
Merrick, R. L.
Ritchie, W. P.

MISSISSIPPI (1)

JACKSON
Neill, C. L.

MISSOURI (15)

KANSAS CITY
Carmichael, F. A., Jr
Coburn, D. F.
Forsythe, R. W.
Lewis, R. C.
Teachenor, F. R.
Weiford, E. C.
Williamson, W. P.
ST. LOUIS
Furlow, L. T.
Hawkins, G. L., Jr.
Klemme, R. M.
Palazzo, F. A.
Roulhac, G. E.
Schwartz, H. G.
Smolik, E. A.
Woolsey, R. D.

MONTANA (1)

GREAT FALLS
Johnson, A. C.

NEBRASKA (4)

LINCOLN
Gogela, L. J.
OMAHA
Finlayson, A. I.
Keegan, J. J.
Muehlig, W. A.

NEVADA (1)

RENO
Mack, E. W.

NEW HAMPSHIRE (1)

HANOVER
Heyl, H. L.

NEW JERSEY (6)

JERSEY CITY
Winokur, G. L.

NEWARK
Ehrlich, W.
Somberg, H. M.
PLAINFIELD
Fitch, T. S. P.
SOUTH ORANGE
Buermann, A.
TRENTON
Scheuerman, W. G.

NEW MEXICO (2)

ALBUQUERQUE
Miller, L. J.
LAS VEGAS
Allen, S. S.

NEW YORK (53)

ALBANY
Burklund, C. W.
Campbell, E. H.
Whitfield, R. D.
BINGHAMTON
Kane, J. W.
BRONX
Krueger, E. G.
BROOKLYN
Browder, J.
Corradini, E. W.
Freedman, H.
Kaplan, H. A.
Rosenberg, M.
BUFFALO
Beswick, W. F.
Graf, C. J.
Hamby, W. B.
Zoll, J. G.
CANANDAIGUA
Liu, C. T.
FLUSHING
Siris, J. H.
JACKSON HEIGHTS
Wigderson, H.
MANHASSET
Shepard, J. W.
NEW YORK
Bridges, T. J., Jr.
Campbell, J. B.
Cohen, I.
Cramer, F.
Daniels, J. T.
Davidoff, L. M.
Ebin, J.
Echlin, F. A.
Feiring, E. H.
Gang, K. M.
Gross, S. W.
de Gutiérrez-Mahoney, C. G.
Hoen, T. I.
Kaplan, A.
MacLean, J. A.
Mount, L. A.
Negrin, J., Jr.
Oljenick, I. W.
Parsons, H.
Pool, J. L.
Ransohoff, J.
Ray, B. S.
Scarff, J. E.
Schlesinger, B.
Schlesinger, E. B.
Stellar, S.
Stookey, B.
Strully, K. J.
Tarlov, I. M.
Wingebach, W.
ROCHESTER
Smith, F. P.
Van Wagenen, W. P.
SCHENECTADY
Mason, T. H.
SYRACUSE
Ecker, A.
Peacher, W. G.

NORTH CAROLINA (9)

ASHEVILLE
Hillier, W. F., Jr.
CHAPEL HILL
Heusner, A. P.
CHARLOTTE
Bellows, R. T.
Pitts, W. R.
DURHAM
Odom, G. L.
Woodhall, B.
GREENSBORO
Ames, R. H.
WINSTON-SALEM
Alexander, E., Jr.
Jeffreys, E. O.

OHIO (29)

AKRON
Hurteau, E. F.
Weinberger, L. M.
CANTON
Boyer, F. C.
CINCINNATI
Chambers, W. R.
Evans, J. P.
Hunter, C. R.
Lotspeich, E. S.
Mayfield, F. H.
Meirowsky, A. M.
Osher, D.
CLEVELAND
Braden, S.
Bunts, A. T.
Elkins, C. W.
Gardner, W. J.
Grino, A.
Nosik, W. A.
Shannon, E. W.
Wolkin, J.
COLUMBUS
Abbott, K. H.
Gay, J. R.
LeFever, H. E.
DAYTON
Hollister, N. R.
Weaver, T. A., Jr.
TOLEDO
Booth, G. T.
Earnest, F., III
Hopple, T. L.
Schnitker, M. T.
YOUNGSTOWN
Bernstein, L. L.
Turner, O. A.

OKLAHOMA (7)

OKLAHOMA CITY
Herrmann, J. D.
Huff, T. J., Jr.
Lisle, A. C., Jr.
Rix, R. A., Jr.
Wilkins, H.
TULSA
Hayne, R. A.
Stowell, A.

OREGON (4)

PORTLAND
Davis, E. W.
Kloos, E. K.
Livingston, K. E.
Raaf, J.

PENNSYLVANIA (25)

ARDMORE
Olsen, A.
BETHLEHEM
Erb, H. R.
ERIE
Kinal, M. E.
HARRISBURG
Markley, G. M.
PHILADELPHIA
Austin, G. M.
Brady, J. A.
Fay, T.
Grant, F. C.
Groff, R. A.
Jaeger, J. R.
Nulsen, F. E.
Scott, M.
Shenkin, H. A.

Spitz, E. B.
Taeffner, J. H.
Whiteley, W. H., III
Wycis, H. T.

PITTSBURGH
Bragdon, F. H.
Grunnagle, J. F.
Koskoff, Y. D.
Lindstrom, P. A.
Rowe, S. N.

READING
Johnson, H. C.

SAYRE
King, A. B.

SCRANTON
Weiss, E. K.

RHODE ISLAND (2)

PROVIDENCE
Hamlin, H.
Silver, M. L.

SOUTH CAROLINA (1)

COLUMBIA
Bridgers, W. H.

TENNESSEE (14)

CHATTANOOGA
McCravey, A.

KNOXVILLE
Smith, W. E.
Turney, M. F.

MEMPHIS
Cannon, B. W.
DeSaussure, R. L., Jr.
Gotten, N.
Hawkes, C. D.
Murphey, F.
Raskind, R.
Schultz, E. C.
Semmes, R. E.
Tyrer, A. R., Jr.

NASHVILLE
Cobb, C. A., Jr.
Meacham, W. F.

TEXAS (20)

AUSTIN
La Londe, A. A.

DALLAS
D'Errico, A.
Weary, W. B.
Wilson, C. M.
Woolf, J. I.

EL PASO
Jones, W. A.

FORT WORTH
McKinney, W. W.
Rehfeldt, F. C.

GALVESTON
Snodgrass, S. R.

HOUSTON
Ashkenazy, M.
Bradford, F. K.
Ehni, G.
Greenwood, J., Jr.
Jones, J. R.
Pollard, C., Jr.
Robertson, R. C. L.

McKINNEY
Kern, C. E.

SAN ANTONIO
Munslow, R. A.

TEMPLE
Walker, J. C.

WACO
Steelman, H. F.

UTAH (2)

SALT LAKE CITY
Powell, C. B.
Wright, S. A.

VERMONT (2)

BURLINGTON
Donaghy, R. M. P.
Wallman, L. J.

VIRGINIA (9)

CHARLOTTESVILLE
Crutchfield, W. G.
Martinez-G., J. de D.

NORFOLK
Thomson, J. L.

PORTSMOUTH
Clare, F. B.

RICHMOND
Meredith, J. M.
Troland, C. E.
Ulmer, J. L.

ROANOKE
Tice, W. P.
Weaver, E. N.

WASHINGTON (7)

SEATTLE
Foltz, E. L.
Haven, H.
Klemperer, W. W.
MacKay, H. J.
Stafford, D. E.
Ward, A. A., Jr.

TACOMA
Robson, J. T.

WEST VIRGINIA (3)

BLUEFIELD
Gage, E. L.

CHARLESTON
Wilson, A. A.

HUNTINGTON
Holbrook, T. J.

WISCONSIN (8)

MADISON
Erickson, T. C.
Suckle, H. M.

MILWAUKEE
Cleveland, D.
Kiefer, E. J.
Levin, J. D.
Maxwell, H. P.
Mufson, J. A.

NEENAH
Quade, R. H.

IN MILITARY SERVICE (4)

Hayes, G. J. (Army)
Luce, J. C. (Navy)
Risteen, W. A. (Navy)
Schultz, A. L. (Navy)

TERRITORY OF HAWAII (3)

HONOLULU
Bennett, T. S.
Cloward, R. B.
Lowrey, J. J.

PUERTO RICO (2)

SAN JUAN
Guzman-Lopez, L. R.
Rifkinson, N.

BRAZIL (1)

BELO HORIZONTE
Albernaz, J. G.

DOMINION OF CANADA (11)

HALIFAX, N. S.
Stevenson, W. D.

MONTREAL, QUE.
Bertrand, C. M.
Cone, W. V.
Elvidge, A. R.
Parenteau, A.
Penfield, W.

TORONTO, ONT.
McKenzie, K. G.

VANCOUVER, B. C.
Fowler, W. J.
Lehman, P.
Turnbull, F.

WINNIPEG, MAN.
Parkinson, D.

CUBA (1)

HAVANA
Garcia-Bengochea, F.

MEXICO (3)

MEXICO, D. F.
Beltran Goni, P.
Cardenas, Y C., J.
Sanchez-G., M.

REPUBLIC OF PANAMA (1)

PANAMA CITY
Revilla, A. G.

PHILIPPINE ISLANDS (1)

MANILA
Reyes, V. A.

TURKEY (1)

ISTANBUL
Berkay, F. M.

VENEZUELA (1)

CARACAS
Castillo, R.

NOTICES

AMERICAN ACADEMY OF NEUROLOGY

The American Academy of Neurology is again offering special courses in various aspects of neurology. They will be given at the Edgewater Beach Hotel, Chicago, Illinois, on April 6, 7 and 8, 1953, preceding the fifth annual meeting of the Academy.

One-day courses include Neuropathology, Clinical Electroencephalography, Episodic Disturbances of the Nervous System, Clinical Neuro-ophthalmology, Language Disabilities, Brain Tumors, and Neuro-anatomy. A two-day course is offered in Neuroroentgenology because of the wealth of material.

These courses are oriented toward practical clinical use. The faculty has been selected for recognized leadership in their field and for their teaching ability.

Fees are moderate and many courses offer atlases, loan-boxes of slides, syllabi, discussion sessions, etc. Details can be obtained by writing Mrs. J. C. McKinley, 19 Millard Hall, University of Minnesota, Minneapolis, Minnesota.

GERMAN SOCIETY OF NEUROSURGERY

The German Society of Neurosurgery, the German Society of Neurology, and the German Society of Neurologists and Psychiatrists will hold a combined meeting in Munich, August 26–29, 1953. Subjects will be: first day, Technique of Narcosis; second day, Meningitis; third day, Development of Psychiatry in the United States During the Last Three Decades; and fourth day, Psychodiagnostics. The German Neuropathological Society will hold its meeting in Munich, concurrently, August 26–27. Subject: Cerebellum. Further information may be obtained from Dr. Ehrhardt, Univ.-Nervenklinik, Marburg/Lahn, Germany.

PERSISTENT CAROTID-BASILAR ANASTOMOSIS

THREE ARTERIOGRAPHICALLY DEMONSTRATED CASES WITH ONE ANATOMICAL SPECIMEN

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(Received for publication September 22, 1952)

THE persistent carotid-basilar anastomosis is a large anomalous vessel joining the cavernous portion of the internal carotid artery to the mid-portion of the basilar artery. It has been called the persistent primitive trigeminal artery because of its relationship to the gasserian ganglion, especially in early fetal life.

The presence of a carotid-basilar anastomosis such as is seen in the 3 cases here presented is caused by the persistence of a vessel prominent in very early development which is ordinarily obliterated when the embryo has reached the 14 mm. stage. Our understanding of how this comes about is based on the work of Padget[13] who first identified this "primitive trigeminal artery" as a branch of the internal carotid in a 20 somite (3 mm.) human embryo. It is in this stage the principal blood supply to the plexus of vessels which form the paired longitudinal neural arteries, precursors of the basilar artery, and is apparently instrumental in their development. The formation of the carotid-basilar anastomosis itself has been suggested by Streeter[16] to be related to the precocity of the gasserian ganglion, to which it is constantly related as long as it exists.

As the brain develops, the origin of this anastomotic channel becomes increasingly angulated. With this distortion, with the development of the posterior communicating arteries and later of the vertebrals to supply the basilar artery, and with the eventual interposition of the basal sphenoid cartilage the carotid-basilar anastomosis disappears. Figs. 1 and 2 illustrate several phases in the history of this artery. The close relationship to the gasserian ganglion is maintained in all recorded instances of persistence of this vessel.

REVIEW OF THE LITERATURE

Since the original illustrated case report of Quain in 1844[14] of a "remarkable branch" between the internal carotid artery and the basilar artery, several authors have described additional cases. The literature is summarized in Table 1. It will be noted that 9 of the anastomoses occurred on the right side and 8 on the left. In 8 of the 16 anatomical specimens, the

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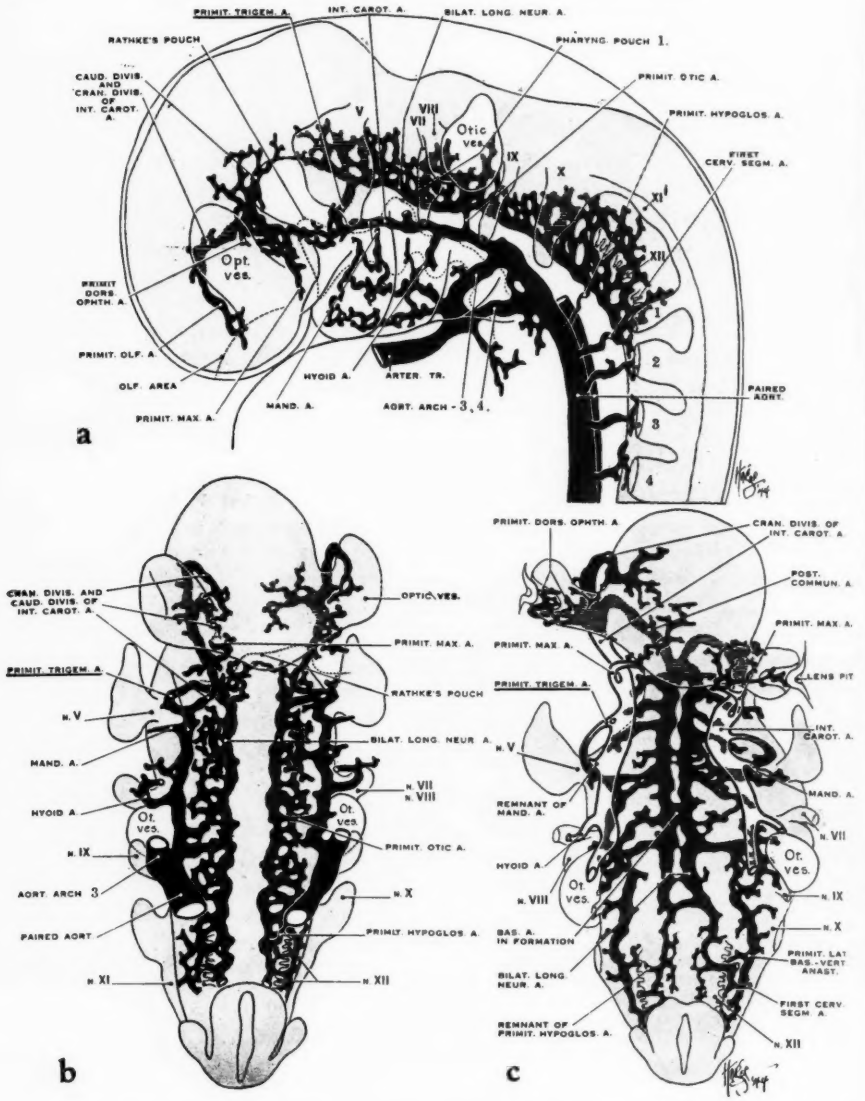

FIG. 1. This illustration and Fig. 2 were assembled by Padget from her original illustrations.[1] The artery under consideration is underlined. (a & b) A 4 mm. embryo showing how the strong trigeminal branch of the internal carotid artery supplies the bilateral longitudinal neural arteries which precede the basilar artery. By the 6 mm. stage (c), the posterior communicating arteries have usually formed to constitute the anterior supply to the neural arteries which are consolidating into the basilar artery. As a result the trigeminal arteries have dwindled.

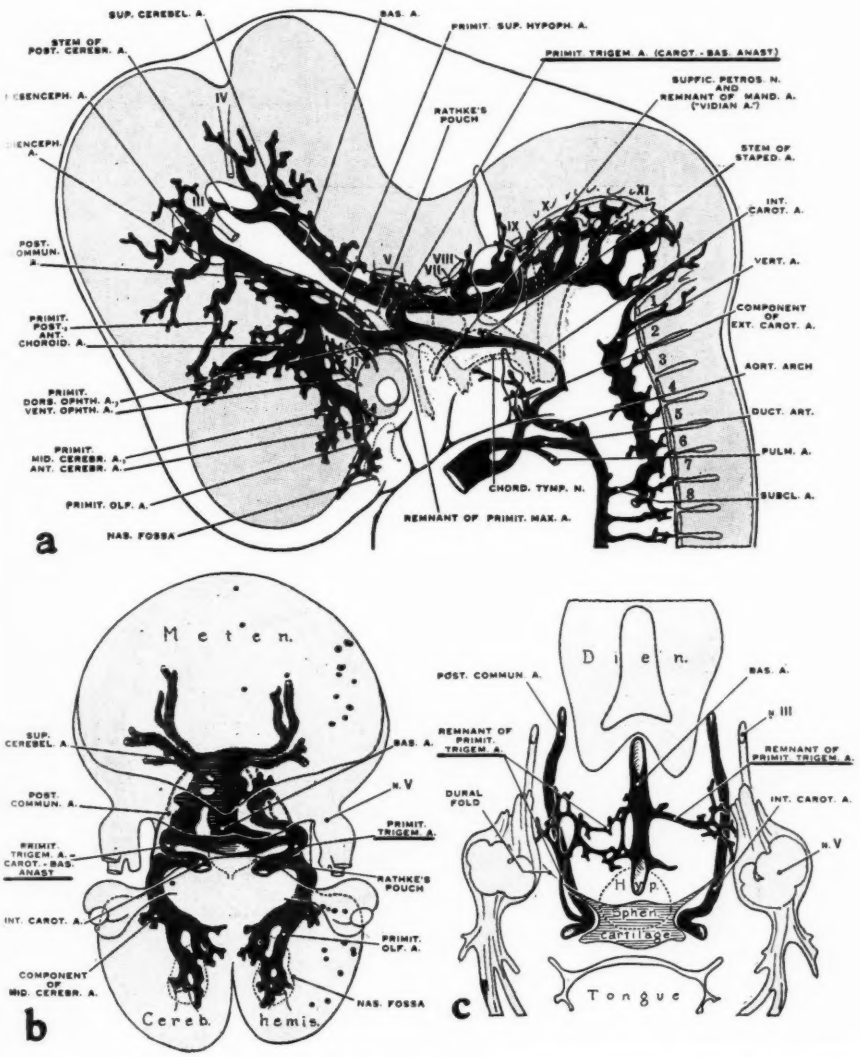

FIG. 2. (a & b) Lateral and cross section views of two embryos approximately 14 mm. long with an unusually late retention of the trigeminal artery. Although the posterior communicating and vertebral arteries have formed, they are small in part and the trigeminal artery still supplies the basilar artery, constituting a large carotid-basilar anastomosis. Normally, as seen at 23 mm. (c), only variable remnants of this early connecting branch are found.

TABLE 1

Author	Year	No. of Cases	Origin from Carotid		Course of C–B Anastomosis in Relation to the Dorsum Sellae	
			Right	Left	Through	Skirts Lateral Margin
Quain[14]	1844	1		1	1	
Tüngel[23]	1860	1	1		1	
Duret[6]	1874	2	2			2
Tarenieckí[22]	1880	1	1		1	
Flesch[7]	1882	1		1		1
Hochstetter[9]	1885	2	2		2	
Decker[5]	1886	1		1		1
Smith[15]	1909	1		1		1
Oertel[11]	1922	1	1		1	
Hasenjäger[8]	1937	1	1		1	
Sunderland[19,20]	1941					
	1948	3		3	1	2
Sutton[21]*	1950	2	1	1		1

* No anatomical specimen obtained with Sutton's second case.

anomalous vessel perforated the dorsum sellae. In the other 8 it skirted this structure extradurally.

Among the articles on this subject, that of Oertel[11] is notable for its review of the literature until 1922. Oertel's case is interesting because of a co-existing persistent hypoglossal artery coursing through the hypoglossal canal and connecting the right vertebral artery to the right internal carotid artery. The carotid-basilar anastomosis in this case was also on the right side. The persistent hypoglossal artery had previously been described by Batujeff[2], but we know of no other instance of the double anomaly described by Oertel.

Sunderland's articles[19,20] are distinguished by the excellence of their illustrations. They stress the intimate relation of the abnormal vessel to the gasserian ganglion and to the sensory root of the trigeminal nerve.

The possibility of demonstrating the persistent carotid-basilar anastomosis by arteriography is mentioned by Hasenjäger.[8] Sutton,[21] however, is at present the only one to report a case diagnosed by this method. A reproduction of the x-ray accompanies his article.

Sutton emphasizes that the direction of blood flow within the anomaly is from carotid to basilar, as his arteriogram confirms. This is borne out by the three arteriograms here reported. Consideration of the derivation of this vessel should lead one to the same conclusion even without arteriographic confirmation. The anastomosis in earliest embryologic development must conduct blood away from the carotid, for it terminates blindly. With increased blood supply to the longitudinal arteries and later the basilar artery from the posterior communicating and vertebral artery the carotid-basila

anastomosis becomes attenuated and finally obliterated, whereas one should expect the opposite were the blood to flow from basilar to carotid.

INCIDENCE

The incidence of this vessel among routine autopsies and cerebral angiograms is impossible to ascertain. It is probably not always seen at postmortem examination when present. It has been reported as an arteriographic finding only once, despite the large number of individuals subjected to this procedure in the past two decades. We have seen another case demonstrated arteriographically by Sugar.[18] In a discussion of mechanisms by which the basilar artery fills during carotid angiography, and drawing from a personal experience with 1,526 cases, Lindgren[10] failed to mention the primitive carotid-basilar anastomosis.

The literature consists largely of single case reports; incidence is usually not given. The case demonstrated arteriographically by Sutton was the only example he found in nearly 1,000 cerebral angiograms. Sunderland's 3 cases were encountered among 210 autopsies. The 3 cases reported here were found among 582 cerebral angiograms examined personally by the authors.

CASE REPORTS

Case 1. J.H.H. 566865. J.L., a 46-year-old white married male school teacher, had been feeling listless for about 6 months. Two months before entry he had had sudden onset of headache accompanied by left-sided weakness, poor coordination and thick speech. He was hospitalized in another institution for 10 days where left hemiparesis was noted. One month before entry here he had a left-sided convulsion. Left-sided weakness persisted, and 2 weeks before entry headache recurred and persisted until admission.

Examination and Course. Significant findings were slight confusion, memory deficit, and left hemiparesis. X-ray of the skull was normal. Right carotid angiogram

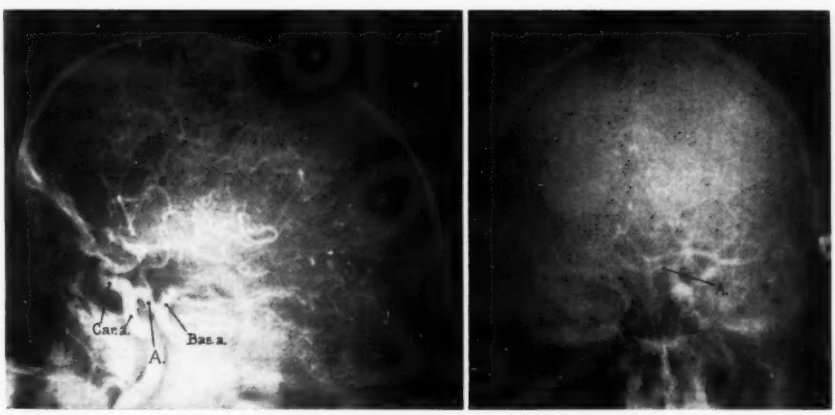

FIG. 3. *Case 1.* Lateral and anteroposterior cerebral arteriograms. (A) Anastomosis.

revealed slight displacement of the anterior cerebral arteries to the left, and a persistent right carotid-basilar anastomosis (Fig. 3). Ventriculogram revealed a tumor mass in the right parietofrontal region.

A right frontal craniotomy and partial right frontal lobectomy were done. Pathological diagnosis: Glioblastoma multiforme.

The patient was discharged on the 14th hospital day. He died at home 2 months later. No autopsy was performed.

Case 2. J.H.H. 596598. I.S., a 54-year-old white married male janitor, was well until the day of entry. Just before noon, he noted sudden onset of severe generalized headache, worst in the region of the occiput. He felt light-headed and collapsed to the floor, but very soon was able to sit up. He vomited once. He seemed confused.

Examination and Course. Significant findings were semistupor and a stiff neck. Lumbar puncture revealed grossly bloody CSF. Emergency right carotid angiography revealed a persistent right carotid-basilar anastomosis (Fig. 4).

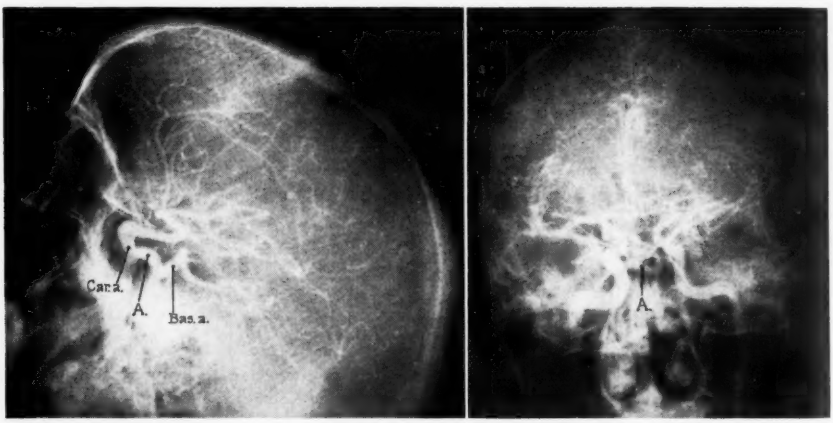

FIG. 4. *Case 2.* Lateral and anteroposterior cerebral arteriograms. Note bilateral filling obtained by compression of the opposite carotid during injection of diodrast. (A) Anastomosis.

On the 2nd hospital day the patient became very irrational. On the 3rd day his temperature rose to 102°F. The pulse became fast. He lapsed into coma, and died shortly thereafter.

Autopsy disclosed a massive intracerebellar hematoma, and persistent carotid-basilar anastomosis.

Case 3. J.H.H. 588456. E.G., a 32-year-old white married female, had had frequent frontal headaches for as long as she could remember. A year before entry, while at work, she had a sudden, unexplained akinetic major seizure. Two months before entry a second akinetic seizure occurred following which her headache grew worse. At this time she began to experience sudden bouts of foul odor in the nostrils lasting a few seconds and not obvious to people about her. She also began to have attacks of twitching and drooping of the right side of the face.

Examination and Course. Significant findings were drooping of the left side of the

FIG. 5. *Case 3.* Lateral and anteroposterior cerebral arteriograms. (A) Anastomosis.

face, slight increase in the reflexes of the right arm, slight weakness of grip on the right, and right hemihypaesthesia with inattention phenomena. EEG showed a diffuse abnormality with slight suggestion of left temporal focal spiking. Left carotid arteriogram revealed a persistent carotid-basilar anastomosis (Fig. 5).

She was discharged as a case of undiagnosed disease manifest by diffuse neurological signs and syncopal attacks.

GROSS ANATOMY OF THE SPECIMEN

The death of Case 2 gave us the opportunity to obtain the portion of the base of the skull containing the anomalous vessel (Fig. 6). The origin of the artery is from the cavernous portion of the left internal carotid. At the point of its origin the caliber of this vessel is almost equal to that of the cavernous portion of the internal carotid artery. The vessel proceeds directly caudally in the cavernous sinus and lies in lateral juxtaposition to the oculomotor nerve and medial to the first division of the trigeminal nerve, the medial portion of the gasserian ganglion, and the sensory root of the trigeminal nerve. As the artery leaves the cavernous sinus it emerges through an opening formed by a groove in the posterior petrosal process whose roof is the petroclinoid ligament. At this point the abducens nerve lies directly beneath it. The course now becomes almost transverse, joining the midportion of the basilar artery over the clivus. Superior to this junction the size of the basilar artery is over twice that of the portion lying between the anastomosis and the vertebral arteries.

Both posterior communicating arteries were very small in caliber, but were patent. The circle of Willis was unfortunately destroyed when the brain was sectioned so that it cannot be reproduced here. An artist's drawing of

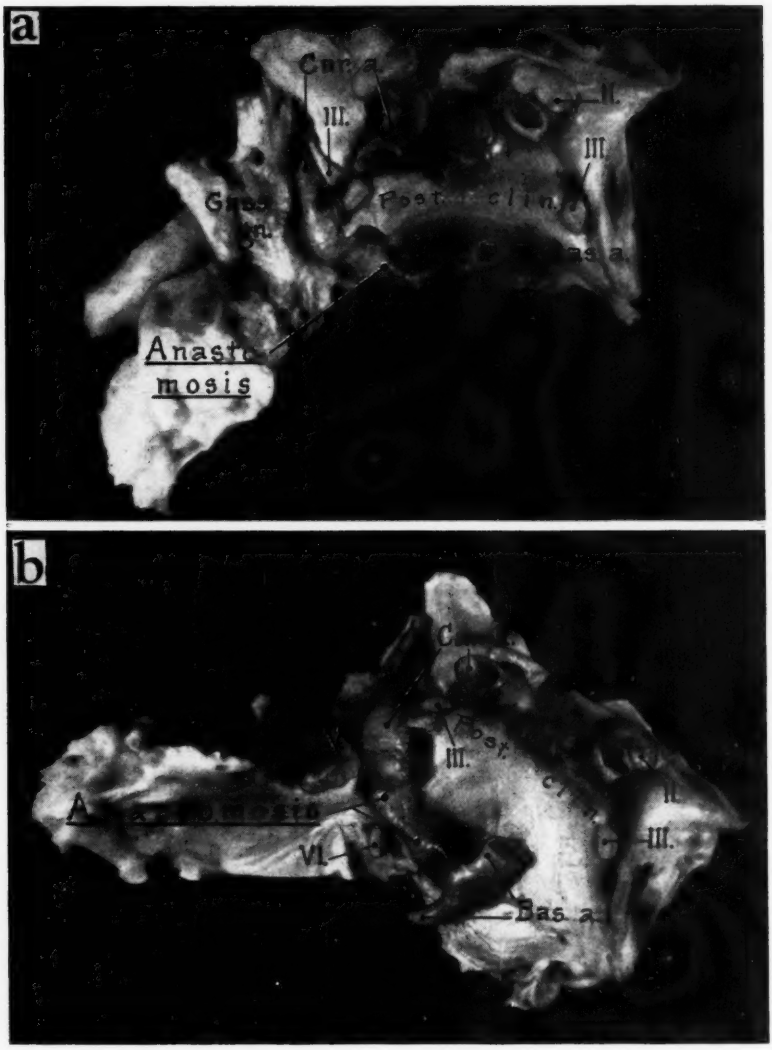

FIG. 6. *Case 2.* (a & b) Photographs of the anatomical specimen. The relationship of the anastomosis to cranial nerves III, V and VI should be noted. Cranial nerve IV has been avulsed.

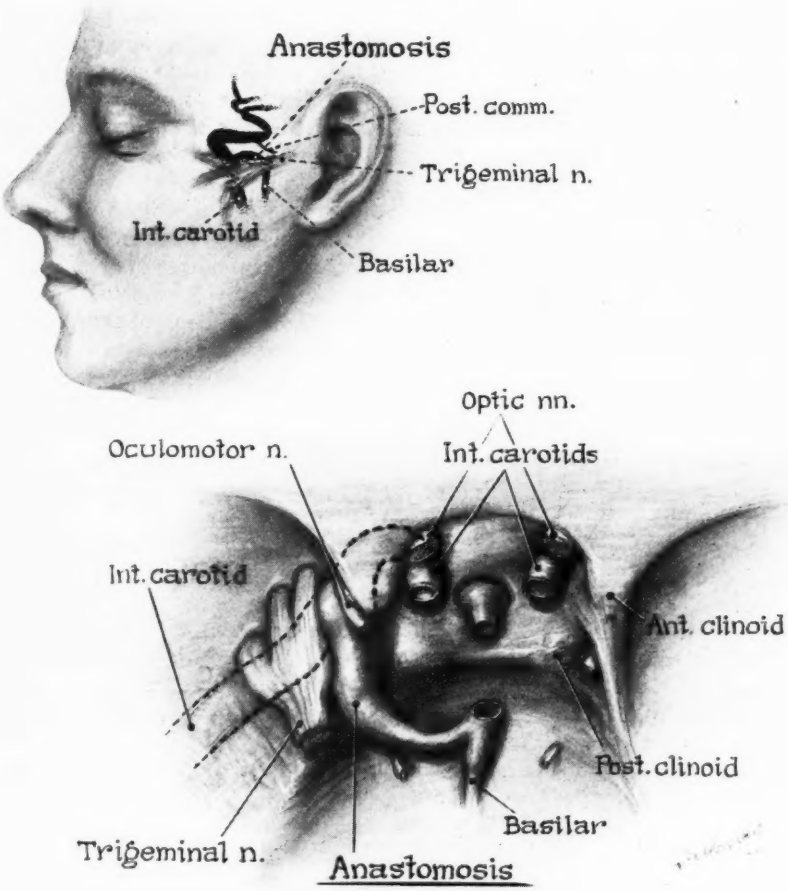

FIG. 7. *Case 2.* Drawing of the anatomical specimen. The position and relationship of the anastomosis are more apparent than in the photograph.

the specimen pictures more clearly than the photographs the relationships of the artery (Fig. 7).

DISCUSSION

A knowledge of variations in the circle of Willis is necessary in the interpretation of cerebral angiograms. The anomaly under consideration is an unusual one, but our experience suggests that it is not as rare as has been supposed. The arteriographic configuration of the persistent carotid-basilar

anastomosis is almost constant, and attention should be directed to the search for this vessel in any angiogram in which the posterior cerebral and basilar arteries are outlined by the carotid injection of radiopaque dye.

The close anatomical relationship of the anastomosing vessel to the sensory root of the 5th cranial nerve assumes salience in operations on or near the gasserian ganglion. An encounter with the persistent carotid-basilar anastomosis during such surgery could be hazardous.

Dandy[3] suggested that pressure on the sensory root of the 5th nerve by an S-shaped tortuosity of the basilar artery can sometimes be responsible for trigeminal neuralgia. The carotid-basilar anastomosis creates similar pressure on the root. Sunderland[20] and Sutton [21] commented on this, but in their cases there was no record of dysfunction of the 5th nerve. It is conceivable that an arteriosclerotic plaque in the vessel might compress the nerve, but no such case has been recorded. Among the cases reported here a review of the past histories failed to disclose any evidence of 5th nerve involvement.

As shown in our specimen the aberrant vessel also bears close relationship to the oculomotor nerve, the trochlear nerve and the abducens nerve in the cavernous sinus. In this case there was no history of extraocular palsies or pupillary abnormalities.

In our specimen the posterior communicating arteries and the basilar artery caudal to the anastomosis are small, and the principal blood supply of the basilar artery was furnished by the anastomotic channel. Sunderland, Altmann,[1] and Oertel, observed that in the presence of a persistent trigeminal artery the vertebral artery on that side or both sides may be considerably reduced in size. Whether failure in development of the vertebral arteries can influence the persistence of the carotid-basilar anastomosis, or whether the presence of the anastomosis prevents development of the vertebral arteries one cannot say. In any event, atresia of the vertebral arteries is compensated for by the presence of this anomaly.

Oertel states that branching points of presegmental arteries in the embryo may remain as small widenings of the carotid arteries or leave a scar at their points of obliteration. Padget[12] has shown that among individuals with cerebral aneurysms, the circle of Willis shows a primitive state of development in about 50 per cent of cases. Dandy,[3] on the basis of Padget's work, was of the opinion that congenital cerebral aneurysms arise from weakened points representing the site of incompletely involuted embryonic vessels. Sugar[17] mentioned that the relatively common aneurysms of the cavernous portion of the internal carotid artery may well arise from just such a weak point representing the site of the obliterated carotid-basilar anastomosis. This seems a wholly reasonable interpretation.

It is not unlikely that weakening of the wall of the carotid artery at the site of the obliterated carotid-basilar anastomosis accounts for the predilection of the carotid to rupture within the cavernous sinus with resulting arteriovenous fistula. Dandy and Follis[4] demonstrated congenital weakness in the wall of the internal carotid artery with loss of the elastic coat at the

location of the fistula in a case which was precipitated by trauma. In a spontaneous case of carotid-cavernous fistula they found two openings in the wall of an aneurysm within the sinus.

SUMMARY

The embryologic origin of the persistent carotid-basilar anastomosis is described. The literature pertaining to this vessel is cited and 3 additional cases, all demonstrated arteriographically, are presented. The gross anatomy of a specimen obtained post mortem in one of these 3 cases is described in detail. Some features of possible interest are discussed.

The authors are greatly indebted to Mrs. Dorcas Hager Padget for her help and advice, especially with reference to the embryology of the anomaly and the illustration of this specimen. They also wish to thank Dr. David Clark, who made this study possible, and Mr. Leon Schlossberg, whose drawing greatly clarifies the relationships of the vessel under discussion.

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THE PATHOLOGY OF HUMAN STRIATED MUSCLE FOLLOWING DENERVATION*

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(Received for publication October 1, 1952)

THE neuromuscular unit forms a physiological mechanism which can be evaluated clinically only by observations on the muscle component. Following severance of peripheral nerves, all parts of the neuromuscular unit degenerate. Specifically, the structures involved are the nerve fibers peripheral to the injury, the motor end-organs and the innervated muscle fibers. When proper recovery fails to occur it usually is assumed that some failure of nerve regeneration has developed, inasmuch as the nerve was the structure that was injured. Such an assumption would not appear warranted, however, since a failure of regeneration on the part of any one of the component structures forming the neuromuscular mechanism could equally well explain the failure of recovery as observed clinically following peripheral nerve injuries. Other factors in addition to the neuromuscular unit, that may impair full functional recovery and that should be thought of as possibly contributing to the picture, are ligament change and joint stiffening.

In a previous study[8] concerned with the relative capacities of regeneration for the various components of the neuromuscular unit following peripheral nerve injury, evidence was presented showing a correlation between the loss of muscle cells and fibrosis on one hand and the faulty recovery of the neuromuscular unit following long delayed procedures of nerve repair on the other hand. The loss of muscle cells and development of fibrosis progressed at a rate roughly proportional to the duration of the atrophic and degenerative changes. On the other hand, after anastomosis of the peripheral nerves following peripheral nerve injuries, no failure in the regeneration of the nerve endings or end-organs was observed, even after prolonged intervals, that could explain adequately the faulty recovery found. The loss of muscle fibers and the mechanical fixation of fibrosis, therefore, were associated with the faulty recovery of the neuromuscular unit after long delayed procedures of nerve repair.

Excellent reviews on peripheral nerve regeneration[14] are available. Their emphasis, however, has been upon the factors at the point of injury and in the peripheral stump or end-organ, which delay the regeneration of nerve

* The work described in this paper was done under a contract (OEM cmr 109) recommended by the Committee on Medical Research between the Office of Scientific Research and Development and the University of California.

fibers and the proper innervation of muscles. In general, the attention of clinicians and investigators has been focused on this phase of the problem. For example, in Young's[14] classic review, great stress was laid upon those factors that impede the regeneration of peripheral nerves and their successful re-innervation of muscle. The importance of muscle in the functional recovery of the neuromuscular unit was barely mentioned.

Aside from the delay in the regeneration of fibers from the central stump to the peripheral stump after peripheral nerve injury and the distance the fibers must regenerate down the peripheral stump before reaching the muscles, Holmes and Young[6] have stressed the necessity of an increase in the diameter and medullation of the regenerated fibers before their functional maturation is achieved. They also have pointed out that further delay is occasioned by the re-innervation of muscle through old, residual end-organs or the formation of new end-organs.

In a more recent contribution, Sunderland[12] has reviewed previous studies concerned with the rate of regeneration of peripheral nerves and added evidence to show that it diminishes progressively as the distance from the cell body increases.

Although these factors may delay the re-innervation for weeks and months, Holmes and Young[6] also pointed out that the power of regeneration remains essentially undiminished in peripheral nerves over long periods. Nevertheless, Young has wisely advocated early intervention.[5,14,15]

In the excellent review of muscle by Tower,[13] the irreversible changes consequent to atrophy and denervation in muscle are dealt with in little more than one paragraph on "Fibrous Transformation." Even here it is emphasized that this change occurs very slowly (requiring more than a year) and the possible role of fibrosis seriously impairing muscle function at earlier stages is not considered.

Much work has been done on the degeneration and regeneration of nerves, muscles and to a less extent of motor end-organs. Apparently, however, no study correlating all three component parts of the neuromuscular mechanism has been made with the object of evaluating their relative powers of regeneration, or of determining which component possesses the least capacity of regeneration.

Holobut and Jalowy[7] correlated the changes in nerve, motor endplates and muscle occurring during degeneration and regeneration, with the functional state of the neuromuscular unit as determined by measurements of chronaxie. Although of great interest in correlating morphological and functional changes, it adds little to our knowledge with respect to the relative powers of regeneration possessed by the various components of the neuromuscular unit.

In an effort to determine whether or not the loss of contractile units and the progressive fibrosis of muscle might account for the failure of recovery in man as was found in dogs,[8] a relatively large series of biopsies from human

muscle denervated over various periods following peripheral nerve injuries have been studied.*

MATERIAL AND METHODS

The study is based on 134 muscle biopsy specimens from 120 patients. The great majority of biopsies were obtained from 3 to 9 months after nerve injury. Periods of denervation, however, varied from 1 day to 26 years. Details of the type and severity of the nerve injuries studied and of the distribution of the muscle biopsies among the peripheral nerves principally involved are shown in Table 1.

TABLE 1

Type and severity of the nerve injuries studied and the incidence of the peripheral nerves principally involved

	Nerve Injury	Number of Biopsies	Number of Patients	Nerve Injured	Number of Biopsies	Number of Patients
Complete Severance	Clean Wound	74	68	Ulnar	47	41
	Infected Wound	20	19	Radial	25	25
Incomplete Severance	Clean Wound	35	28	Common Peroneal	25	23
	Infected Wound	5	5	Others	37	31
Total		134	120	Total	134	120

To avoid distortion, the biopsy specimens were stretched over wooden (tongue blade) blocks and held in place by suture. The specimens then were fixed in 10 per cent formalin. All sections were stained with hematoxylin and eosin. In addition, collagen and reticulin, important in the estimation of fibrosis, were stained by the van Gieson and Laidlaw methods. Nerve endings and end-organs were stained by the activated protargol technique (Bodian). This stain gave an excellent picture of the changes occurring in the muscle fibers as well as of the vascular elements and cellular infiltrations.

RESULTS

An analysis of the pathologic data suggests that one should distinguish

* Inasmuch as surgery on peripheral nerves only occasionally permits a biopsy on the muscle denervated by the injury, the difficulties in obtaining this type of material for study should be emphasized. Ordinarily sufficient material for such a study might be collected only over a period of many years, even in large and active surgical centers dealing frequently with peripheral nerve injuries. Consequently, we wish gratefully to acknowledge the splendid cooperation of the neurological surgeons of the Army, Navy and civilian life who have made this study possible. This investigation has formed the basis of a National Research project supported by the O.S.R.D. and facilitated by the cooperation of the Surgeons Generals of the Army and the Navy.

between those injuries in which the severance of the peripheral nerves was complete as opposed to those in which it was incomplete. Although the data on biopsies from patients with incomplete nerve lesions were insufficient to permit any final conclusion, it is of interest that irrespective of the time interval between injury and operation they showed atrophic and degenerative changes ranging from slight to moderate degrees only, and as a rule no fibrosis, or at the most only slight and early fibrotic changes. From this it would appear that preservation of some intact nerve fibers in peripheral nerve injuries is an important deterrent to both atrophy and fibrosis of muscles. On the other hand, this result might merely reflect the fact that

TABLE 2

Summary of pathologic data on muscle biopsies from patients with peripheral nerve injuries, in which there was a complete severance of the innervating nerve and no infection, and correlation with the time intervals between the injury and the operation

	Interval (Days between Injury and Operation)					
	1–180		181–360		360–more	
	No.	%	No.	%	No.	%
Atrophy						
None	0	0	0	0	0	0
Slight	23	48	11	27	1	14
Moderate	23	48	18	45	2	29
Advanced	2	4	11	28	4	57
Degeneration						
None	11	27	5	17	1	20
Slight	19	46	9	30	0	0
Moderate	7	17	7	23	0	0
Advanced	4	10	9	30	4	80
Fibrosis						
None	18	45	7	21	0	0
Slight	19	48	16	49	2	33
Moderate	2	5	7	21	2	33
Advanced	1	2	3	9	2	33

large portions of the muscle were either not denervated or only temporarily denervated.

The data on peripheral nerve injuries with infected wounds were inadequate to permit an analysis since in most instances we were not provided with information as to whether the infected area involved the site of the biopsy or only the site of the nerve injury at a higher level.

The peripheral nerve injuries with complete interruption and clean wounds permitted a more detailed analysis of the pathologic data with respect to the time interval between the injury and operation. Even in this group, however, the data were not adequate to permit a comparative analysis of the results in different peripheral nerves. The pathologic evaluation of the atrophic, degenerative and fibrotic changes found in the muscles of this

group have been segregated into four categories (no changes, slight, moderate and advanced), and correlated with the intervals of time (taken by 6 month intervals for the first 12 months and from one year on) between the original injury and the final operation (Table 2). In many instances the degree of atrophy, degeneration and fibrosis was found to overlap into two categories, as for example, "slight and moderate" or "moderate and advanced." In these cases both categories were recorded and it is for this reason that the sum of the various pathologic classifications exceeds the total number of biopsy specimens studied.

A more detailed analysis of the pathologic data with respect to the time intervals between the original injury and the operation (every 3 and every 6 months for the first year and from one year on) was made possible (Table 3) by the grouping of the data into two categories, changes of "none-slight" and "moderate-advanced" degrees. This type of analysis has the advantage not only in that it permits a closer correlation of the pathologic changes with respect to time, but also in that it presents the pathologic data in a more simple manner, which serves to differentiate minimal changes from more marked alterations of probable functional significance.

The results presented in Tables 2 and 3 are summarized and their significance is considered below under Discussion.

TABLE 3

Summary of pathologic data on muscle biopsies from patients with peripheral nerve injuries in which there was a complete severance of the innervating nerve and no infection, and correlation with the time intervals between the injury and the operation

	Intervals (Days between Injury and Operation)													
	1–90		91–180		1–180		181–270		271–360		181–360		361–more	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Atrophy														
None-Slight	9	75	14	39	23	48	10	31	1	12	11	27	1	14
Mod.-Adv.	3	25	22	61	25	52	22	69	7	88	29	73	6	86
Degeneration														
None-Slight	9	82	21	70	30	73	14	61	0	0	14	47	1	20
Mod.-Adv.	2	18	9	30	11	27	9	39	7	100	16	53	4	80
Fibrosis														
None-Slight	10	91	27	93	37	93	20	83	3	33	23	70	2	33
Mod.-Adv.	1	9	2	7	3	7	4	17	6	67	10	30	4	67

DISCUSSION

Tower[13] has emphasized the distinction between atrophy and degeneration following the denervation of striated muscles. A review of our findings in a previous study[8] and the results of the present investigation tend to support such a distinction. The marked variation in degenerative changes found in individual cases and the lack of correlation between these changes

and either the atrophic changes or interval from the time of injury would appear to justify such a distinction. On the other hand, as indicated in the results presented in Tables 2 and 3, the degenerative changes were found in a high percentage of the biopsies and, in general, followed, although lagged behind, the atrophic changes. Because of this we have attached more significance to the degenerative changes observed in our material than apparently was attributed by Tower to similar changes in her studies. She states: "The end result in man of continued denervation of muscle is generally conceded to be complete destruction of the muscle tissue, only fibrous tissue remaining. In some cases this destruction takes place extraordinarily rapidly, but usually it is a matter of years. Although the acute degenerative processes undoubtedly contribute to this end, in the experimental animal where I studied it they are of minor significance compared with the much more insidious process of fibrous metaplasia." The high and relatively early incidence of degenerative changes in our human material and the suggestive correlation between such changes and the advancement of fibrosis in individual cases make us feel that the role of degeneration in denervated muscle is not inconsiderable. It would appear that a distinction should be drawn in this respect between experimental denervation studies such as those of Tower[13] and our own,[8] and the present study in which such unknown variables as ischemia, immobilization etc. may have played a part and contributed to the greater degree of degeneration found in our human material. If Tower used the term "fibrous metaplasia" to indicate a fibrous transformation of atrophic muscle fibers, the distinction suggested should be emphasized inasmuch as we believe a fibrous replacement by the endomysium and by infiltrating mesenchymal cells is responsible in large part for the fibrosis observed in our human material.

Our results support the evidence of Tower that atrophy appears to be the principal change associated with fibrosis in muscle. The close relation-

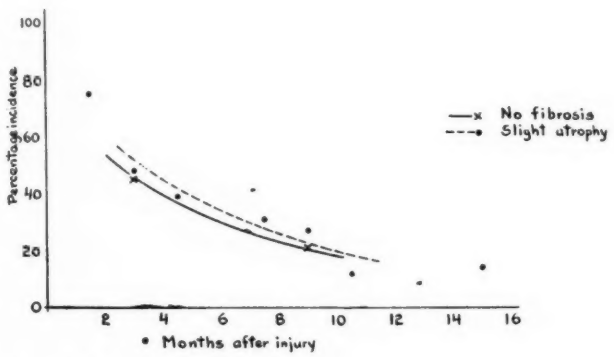

FIG. 1. The relationship between the percentage-incidence of slight atrophy and no fibrosis in human muscles at various periods following denervation as a result of peripheral nerve injuries.

FIG. 2. The relationship between the percentage-incidence of moderate atrophy and slight fibrosis in human muscles at various periods following denervation as a result of peripheral nerve injuries.

ship between atrophy and fibrosis at all stages and time intervals may be clearly demonstrated by graphic methods, if the results are plotted with time represented on the abscissa and the percentage incidence of pathologic involvement (atrophy and fibrosis) of the muscles on the ordinate. Thus, in Fig. 1, it is apparent that the curve representing the percentage of biopsies showing "no" fibrosis parallels the curve for the biopsies showing "slight" atrophic changes.

In Fig. 2 it may be seen that the curve of the biopsies showing "slight" fibrosis closely follows the curve for biopsies exhibiting "moderate" atrophy. Again Fig. 3 indicates that the curve for the biopsies showing "advanced" atrophy follows the curve for the biopsies with "moderate" and "advanced" fibrosis. These results are in close agreement with the earlier results obtained experimentally on dogs.

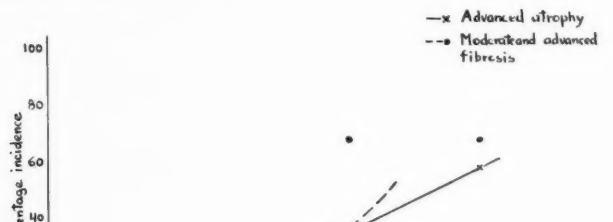

FIG. 3. The relationship between the percentage-incidence of advanced atrophy and moderate and advanced fibrosis in human muscles at various periods following denervation as a result of peripheral nerve injuries.

By plotting several of the curves for atrophy and fibrosis from the data of Tables 2 and 3 on the same graph (Fig. 4) certain interesting relationships become apparent. Thus, in Fig. 4 if the curve for "moderate" and "advanced" atrophy combined is plotted from the data in Table 3, it may be seen to be the reciprocal of the curve for "no" and "slight" atrophy. The point at which "moderate" and "advanced" atrophy combined occurs in a higher percentage of the denervated muscles than "no" and "slight" atrophy, may be seen to come approximately at the 3rd month after injury. Considering the close relationship between muscle atrophy and fibrosis, as indicated above (Figs. 1, 2, and 3), it is not surprising to find that the curve for "moderate" and "advanced" fibrosis begins to swing up within 2 months

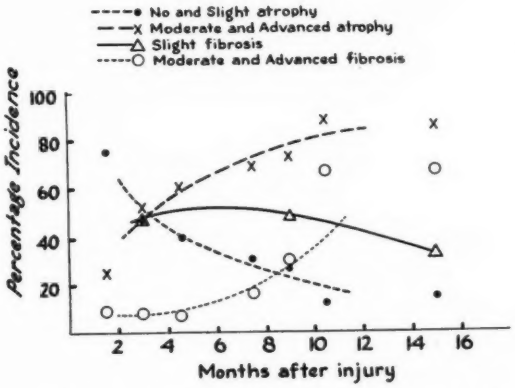

FIG. 4. The percentage-incidence of various stages of atrophy and fibrosis in human muscles at different periods following denervation as a result of peripheral nerve injuries.

of this same period. This relationship suggests that following complete severance of peripheral nerves, surgery should not be delayed beyond the time that might reasonably be expected to provide a successful re-innervation of the involved muscles within a period of 3 or 4 months. Since re-innervation of muscles may not be expected for considerable periods after reparative surgery, depending upon such factors as the distance of the muscle from the point of injury, whether or not the wound was infected (see Results) and mechanisms tending to delay the successful regeneration of peripheral nerves and re-innervation of muscles,[5,6,12,14,15] it would appear that surgical intervention should not be delayed beyond the 1st month or so following complete peripheral nerve injuries.

Again, it may be observed in this same graph that the interval at which "moderate" and "advanced" fibrosis combined (grouped as presented in Table 3; also see Fig. 3) appear in a higher percentage (50 per cent) of denervated muscles than "slight" fibrosis (data from Table 2; also see Fig. 2) occurs at approximately 11 months after the injury. Incidentally, it is of

interest that the curves of "advanced" atrophy (Fig. 3) and of "moderate" atrophy (Fig. 2) cross near the same point and time. Delay in surgical exploration and repair of completely severed nerves beyond this point would appear to carry serious hazards.

Such a statistical analysis by no means affords a true picture of the wide degree of individual variations to be found in the pathologic changes at corresponding periods following injury. The findings in human muscle verify our earlier observations on the dog[8] in this respect. Because of the wide individual variations to be found, the curves presented in the figures cannot be accepted as more than idealized representations of the average results obtained. Nevertheless, they would appear to possess some statistical significance and value as a guide to practical therapy.

The results of this study verify the experimental observation on dogs[8] in still another respect, namely, that the mechanical fixation of fibrosis is an important factor in the failure of proper functional recovery following peripheral nerve injuries. Even during the late war it would appear that the importance of this factor had not gained proper recognition. As in all previous studies along this line, recent research efforts have been directed in the main on the nerve rather than the muscle component of the neuromuscular

FIG. 5. Peroneus longus muscle (E.M.) 97 days after complete severance of sciatic nerve. The muscle fibers are swollen, have completely lost their cross-striations and take the van Gieson stain to a variable degree. Also, note separation of the muscle fibers and early infiltration of fibrosis. The section was interpreted as showing a moderate degree of atrophy and slight fibrosis. ×120.

unit. In particular, the impairment of the contractile function of muscle in relatively early stages of fibrosis has not been properly emphasized, or even considered in most investigations. Considerable infiltration of collagen between muscle fibers has been observed repeatedly as early as the 3rd month (Fig. 5). Although the pathological physiology of such an intimate investment of the contractile units within muscle by noncontractile elements can be only roughly estimated by pathologic examination, our studies strongly suggest that this process may severely impair the function of the denervated muscle. Further advancement of the fibrotic process progressively accentuates this internal, mechanical fixation of the muscle and correspondingly the ability of the muscle to recover is permanently impaired.

Although the importance of fibrotic changes in muscle as a consequence of peripheral nerve injuries has not been fully recognized or emphasized, practical experience has long led neurological surgeons and others to advocate early reparative surgery. This point was stressed by Brown[2] and others[1,11] well before World War II and again has been admirably emphasized by Spurling[11] in his review of the experience of the Army Medical Corps in the European theatre of operations.

The delaying effect of electrotherapy and physical therapy upon atrophy and fibrosis in denervated muscle would be of great importance if verified. Favorable evidence has been reported by several investigators, but denied by others. The favorable results obtained by Gutmann and Guttmann[4] with electrotherapy in the rabbit have not been confirmed in the cat by Pollock *et al.*[10] who, therefore, concluded that there is a species difference in response to electrotherapy. The latter authors found a small increase in the weight of the electrically treated muscles of the rat as compared with untreated control muscles, but this superior weight did not increase with time, which led them to conclude that the difference "probably may be explained on the basis of generalized lessening of the activity of the cat." They also found "greater numbers of contractures of greater severity occurred in the normal antagonistic gastrocnemius muscle when an extremity, after peroneal nerve section and suture, was treated by electrical stimuli" and "This was also found in the experiment upon the sciatic nerve."

On the other hand, the use of massage and passive motion has given promising results. Chor *et al.*,[3] who found no retardation of atrophy and degeneration of denervated muscles with either electrotherapy or massage and passive movement and no effect upon regeneration with the use of these same measures, nevertheless noted a beneficial effect with massage and passive movement in maintaining the condition of the denervated muscle, which they felt to be of importance in determining the degree of muscle repair and recovery. It was interesting that they obtained better results if a preliminary rest period of 4 weeks was allowed before treatment was started, than if treatment was initiated directly following nerve section and suture.

Pollock *et al.*[9] reported that massage and passive movement in the cat not only retarded the development of contractures, diminished their severity

and facilitated their disappearance, but suggestively accelerated the recovery of voluntary and reflex motion and possibly improved the successful neurotization of the distal segment of a sutured nerve. They, also, found, however, that such therapy did not appreciably modify the amount of atrophy or the histological picture, otherwise, of the denervated muscles they studied.

The beneficial effects of physical therapy, therefore, would appear to be based primarily upon massage and passive movement. Furthermore, such beneficial effects, if verified, apparently are not ascribable to any effect in delaying atrophy of the involved muscles. Their value in delaying or overcoming the effects of fibrosis in denervated muscle would appear to be the crucial point if the studies quoted and our own results are of significance. As to how such an effect might be produced, whether indirectly by improving the vascular supply of the muscle, etc., can not be stated on the basis of available studies. Present evidence, although limited and of doubtful value, nevertheless suggests that the effects of massage and passive movement should be further studied and evaluated in this connection.

CONCLUSIONS

1. Pathologic study of denervated muscle in man suggests that in clean wounds with complete severance of the peripheral nerves a close correlation exists between atrophy and fibrosis in muscles at all stages and time intervals. Specifically, the percentage of muscles exhibiting no fibrosis correlated with the percentage of muscles showing "slight" atrophy and in similar fashion a correlation could be made between "advanced" atrophy and "moderate" and "advanced" fibrosis. These findings suggest that atrophy is the primary factor contributing to fibrosis of muscle. In addition, degenerative changes appeared to be an important contributing factor to fibrosis in denervated human muscle.

2. These studies show that interstitial fibrosis may occur in appreciable degree in muscle as early as 3 months after denervation. This intimate investment of the contractile units within muscle by noncontractile elements was found to progress in proportion to the period of denervation. The findings strongly suggest that this internal, mechanical fixation of the muscle by fibrosis may severely and permanently impair the function of denervated human muscle at relatively early stages.

3. The incidence of "moderate" and "advanced" atrophy in muscles surpassed the incidence (50 per cent) of "no" and "slight" atrophy approximately at the 3rd month following denervation. In view of the demonstrated close relationship between muscle atrophy and fibrosis and the fact that the incidence for "moderate" and "advanced" fibrosis showed a sharp increase within the subsequent 2 months, reparative surgery ideally should not be delayed beyond the time that might reasonably be expected to provide a successful re-innervation of the involved muscle within a period of 3 or 4 months.

4. The incidence of "moderate" and "advanced" fibrosis combined in denervated human muscle surpassed the incidence (50 per cent) of "slight" fibrosis at approximately 11 months after peripheral nerve injuries. It would appear that delay in surgical intervention beyond this point might well result in a poor functional recovery.

5. The results obtained in our pathologic studies on human denervated muscle were found to correspond well with similar experimental observations on dogs with respect to (a) the wide degree of individual variation to be found in the pathologic changes in human muscle at corresponding intervals after denervation, (b) the progression of atrophy, degeneration and fibrosis in denervated human muscle relative to the period of denervation and (c) the association of fibrosis with the functional failure of muscle recovery.

6. Considering the importance of the role played by fibrosis in impairing the functional recovery of muscle after peripheral nerve injuries and because physical therapy in the form of massage and passive movement may delay fibrosis, or at least aid in overcoming its effects in restoring muscle function, a further trial and evaluation of these measures would appear highly desirable.

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TRACHEOTOMY IN NEUROSURGERY

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CRANIAL trepanation and tracheotomy are among the oldest surgical procedures known.[1] It is interesting that reports of the benefit of tracheotomy in the treatment of severe head injuries have only recently appeared.[6,12] At the Hartford Hospital, the department of neurosurgery first became convinced of the value of tracheotomy in 1946. Since then, 35 patients in coma from brain injury or primary brain pathology have been tracheotomized during the critical phase of their illness.

AIRWAY AND PULMONARY COMPLICATIONS

The general nursing staff is incapable of properly maintaining evacuation of the nasopharynx and trachea of blood, secretions, and vomitus, by suction. The most conscientious effort often produces minor nasopharyngeal hemorrhages, edema, and inflammation, while the contents of the trachea are often never reached by the suction tube. Tracheotomy reduces this problem to one within the capacity of even relatively inexperienced nursing personnel. The airway is greatly improved in patients who have labored and stertorous breathing. Smooth gentle respirations of good excursion ensue as soon as the tube is in place and the tenacious secretions are removed.

DYNAMICS

When head and chest trauma are concomitant, tracheotomy is doubly indicated. It is generally agreed[1,15] that in severe thoracic injury with multiple fractures of the ribs, tracheotomy reduces splinting by grunting and voluntary glottic closure caused by pain, and decreases the paradoxic chest excursion. The incidence of the usual sequelae of bronchopneumonia and "wet lung" is greatly lowered by early tracheotomy.

The close dynamic relationship between the intrathoracic and intracranial cavities is dependent mainly on the hydraulic transmission of pressure differentials afforded by the vascular connections between the two areas. In his 14th century textbook of surgery, Henri de Mondeville [11] mentioned the now so-called "Valsalva maneuvre" as a test for dural tears. The same principle is all too often dramatically demonstrated at craniotomy when the patient is allowed to cough or strain. Previous hemostasis may be completely undone by such an occurrence. Pneumocephalus in compound cranial fractures was observed by Chiari[2] to follow sneezing. The frequency with which cerebrovascular accidents are associated with straining is well

TABLE 1

Traumatic cases

Case	Age and Sex	Clinical Status	Indication for Tracheotomy	Survival	Condition on Discharge
1	33 F	Dilated fixed pupil, coma	Severe resp. obstruction	2 days	Autopsy: Basilar skull fract., laceration brain
2	55 M	Dilated fixed pupils, bloody CSF	Laryngeal spasm	2 days	Autopsy: Pontine hemorrhage, basilar skull fract., laceration brain
3	55 M	Deep coma, depressed resp., laceration & contusion brain	Obstruction by bronchial secretions	Living	Aphasia, left hemiparesis
4	46 M	Deep coma, brain extruding from nostrils	Pharyngeal obstruction by hemorrhage & brain	3 days	Autopsy: Basilar skull fract., midbrain & frontal lobe contusion, focal pneumonia
5	55 F	Pulsating exophthalmos, basilar skull fract., A-V fistula, contusion chest	Depression resp. after ligation int. carotid	Survived	Left hemiplegia, emotional instability
6	72 M	Coma, massive cerebral contusions, dislocated shoulder	Depressed resp.	59 days	Autopsy: +++ areas encephalomalacia, focal pneumonia
7	83 F	Dilated fixed pupils, coma	Resp. obstruction	8 days	Skull fract., cerebral hemorrhage & thrombosis
8	51 F	Admitted 12 days after severe head injury. Old subdural removed	Laryngeal stridor, resp. retraction	26 days	Cerebral contusions, severe. Old infarct, left upper lobe & kidney
9	20 F	Deep coma, decerebrate rigidity, dilated fixed pupils	Laryngeal spasm	5 days	+++ petechial hemorrhages throughout brain
10	60 M	Fell on street—comatose, skull fract.	Resp. obstruction	1 day	Op. finding: Epidural, subdural & intracortical hematoma
11	21 M	Coma, decerebrate rigidity	Resp. obstruction	24 days	+++ areas encephalomalacia, nephrosis
12	53 M	Coma, G.I. bleeding	Resp. obstruction	5 days	Cerebral contusion & edema
26	30 M	Coma, bleeding right ear, labored resp.	Resp. depressed	1 day	Autopsy: Basilar fract., +++ brain lacerations
31	5 M	Coma, Cheyne-Stokes resp. Dilated fixed pupil, extensive skull fract.	Poor resp. exchange	6 hours	Autopsy: Edema brain, hemothorax, retroperitoneal hemorrhage
32	54 M	Grand mal seizure after fall, coma, pupils dilated & fixed	Increasing resp. embarrassment	1 day	Acute subdural, massive contusions & swelling
33	17 M	Decerebrate rigidity, bloody CSF, pin-point pupils, stertorous resp.	Resp. obstruction	Living	
34	10 M	Restless coma, hemiplegia, otorhinorrhea, fixed pupil, Cheyne-Stokes resp., extensor spasms	Resp. depression & obstruction	Living	Op. findings: Cerebral contusion & edema

known. It is easy to conceive that the increased fluctuation of intrathoracic pressure produced by partial obstruction of the airway, when transmitted to the intracranial cavity, might seriously aggravate bleeding, especially if venous. Tracheotomy, by reducing these fluctuations of pressure, acts as a safety vent toward stabilization of intracranial pressure.

ANOXIA AND CHEMICAL FACTORS

Cerebral anoxia may produce, aside from primary neuronal death, cerebral edema and increased intracranial pressure, hypertension, and dilatation of cerebral vessels. The accompanying rise in plasma CO_2 is, of course, a powerful vasodilator.[8,10,13,14] There is considerable evidence in animal

TABLE 2

Miscellaneous groups

Case	Age and Sex	Clinical Status	Indication for Tracheotomy	Survival	Condition on Discharge
			Brain Tumors and Abscesses		
13	57 M	Postop. acoustic neurinoma	Resp. depression	18 days	Secondary coronary occlusion, arteriosclerosis
14	57 F	Unresponsive after removal acoustic neurinoma	Resp. depression	6 days	Pontine hemorrhage
15	20 F	Postop. large dermoid tumor of post. fossa	Increasing resp. depression +obstruction by secretion	Survived	
16	55 M	Comatose, 4+ bilat. papilledema	Resp. depression	1 day	Massive glioblastoma left hemisphere
17	44 M	Coma, unilat. pyramidal signs	Resp. depression	8 hrs.	Large metastatic carcinoma left temporal lobe
18	65 M	Postop. meningioma, hyperpyrexia, semicoma	Obstructed airway	8 days	Large bilat. meningioma, thrombosis sup. long. sinus, carcinoma prostate, gastric ulcers, acute pylorospasm
19	46 M	Postop. prefrontal lobotomy; meningitis with brain abscess	Resp. depression	Living	Occasional seizures, psychotic
20	49 F	Coma after removal brain abscess	Resp. depression & obstruction	Survived	Left hemiparesis
30	62 F	Dilated fixed pupil, mental confusion, monoparesis, large glioblastoma removed	Resp. depression	Living	Discharged after 7 weeks; able to walk, still confused
21	37 M	Frontal lobe abscess after sinusitis. Moribund, failed to improve after abscess removed	Severe resp. depression	7 days	Autopsy: Secondary abscess pons
			Cerebrovascular Cases		
22	53 M	Coma, unconscious, resp. depression	Resp. depression	1 day	Autopsy: Massive intracerebral hemorrhage, intraventricular rupture
23	65 F	Coma, dilated fixed pupils, resp. depression	Stertorous breathing	12 days	Autopsy: Infarction of pons
25	42 F	Left hemiparesis, dilated fixed pupil. Carotid ligation done	Resp. depression	Survived	Improved with minimal neur. signs
27	50 M	Drowsiness, hemiparesis. CSF bloody; pressure over 600. Exploration for aneurysm	Cheyne-Stokes resp.	1 day	Autopsy: Ant. communicating aneurysm, massive intracortical hematoma
28	58 F	Left hemiparesis. Mental confusion progressed to coma over several days	Labored resp.	18 days	Basilar artery thrombosis
29	38 M	Comatose, dilated fixed pupil, episode of cyanosis. Subdural hematoma evacuated	Episode of cyanosis & depressed resp.	Survived	Mild motor aphasia
36	44 M	Coma, dilated fixed pupils	Cheyne-Stokes resp. Poor airway	4 hrs.	Intracerebral & intraventricular hemorrhage
			Unclassified Case		
35	34 F	Sudden onset confusion progressing to coma; internal hydrocephalus. Cleared completely. Recurrence 9 mo. later; ant. & post. 3rd vent. explor. negative. Torkildsen op. done	Intermittent airway obstruction, episodes of cyanosis	Living	Possible sarcoidosis (biopsy), waxy flexibility, dilated pupils, facial tremors, conscious but almost completely out of contact

experimentation that the bradycardia and hypertension produced by increased intracranial pressure,[3,4] by causing right atrial and pulmonary hypertension induce pulmonary edema.[7] This tendency would be increased by the negative intrathoracic pressure caused by an obstruction of the airway. That this phenomenon has not been recorded in humans may well be because of the fact demonstrated by Durlacher, Banfield and Bergner[5] that very early autopsy is necessary for the true evaluation of pulmonary edema of less than massive proportions. With prolonged anoxia the cough reflex is depressed and respiration, while initially stimulated, is markedly depressed. Tracheotomy reduces the dead space and tidal air,[1] thereby lowering the dilution factor and allowing a significantly higher partial pressure of O_2 to reach the respiratory epithelium than is possible by the intranasal or mask methods. Elimination of CO_2 is promoted. A #18 polyethylene tube is used for O_2 delivery in this clinic in order to encroach as little as possible in the lumen of the tracheotomy tube.

In this series, there were no cases in which tracheotomy *per se* could be said to have contributed to the cause of death, nor were there any complications to the procedure. Table 1 summarizes 17 cases of brain injuries in which tracheotomy was performed to relieve respiratory embarrassment, and Table 2 includes 18 cases of various other intracranial problems. In those cases in which the patient survived, the wound healed promptly without sequelae. The overall mortality rate was 69 per cent, which is formidable. However, realizing that the mortality in this series might well have been 100 per cent, we believe that tracheotomy may have contributed significantly to the survivals.

CONCLUSIONS

We believe that tracheotomy has a justifiable physiologic and therapeutic basis in the treatment of severe head injury and coma of other origin, when serious embarrassment of respiration is a problem. We are in agreement with Echols *et al.*[6] that many so-called "central respiratory failures" are the result of the vicious cycle set off by mechanical obstruction of the airway in the presence of excessive production of mucus. As pointed out by Jefferson,[9] the balance between life and death in head injury is often a very delicate one, and an irreversible chain reaction of failure of interdependent cell masses may be easily initiated. We feel very strongly that tracheotomy or intubation may often weight this delicate balance in favor of survival if performed before this irreversible chain reaction becomes established.

SUMMARY

1. Thirty-five patients who had tracheotomy performed because of respiratory complications in intracranial pathology are presented.

2. A plea is made for early tracheotomy when respiratory embarrassment complicates cranial injury.

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LEPTOMENINGEAL CYSTS OF THE BRAIN FOLLOWING TRAUMA WITH EROSION OF THE SKULL

A STUDY OF SEVEN CASES TREATED BY SURGERY

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LEPTOMENINGEAL cysts, which result from trauma, are fluid-filled spaces between the pia mater and the arachnoid membrane. The fluid-containing pockets are not true cysts because they are not completely isolated from the subarachnoid space, although they are confined by pia-arachnoid adhesions from free communication with the remainder of the subarachnoid space. Pulsations of the brain are transmitted through the cysts to the inner table of the skull and erosion of the bone ordinarily occurs in a localized area overlying the cysts.

It is the purpose of this paper to present, in summary manner, the case histories of 7 patients with leptomeningeal cyst following trauma and to evaluate the results of surgical treatment. The theory of formation of the cysts will be discussed together with the mechanism of the associated bone erosion. Cysts resulting from congenital malformation, from inflammation and from all causes other than trauma are excluded from this study.

MATERIAL

At the Neurological Institute of New York leptomeningeal cysts are relatively rare in their occurrence. The lesion has been encountered more frequently in recent years since the clinical features have been made known and patients have been referred for neurosurgical treatment. The development of leptomeningeal cysts has been recognized more often also as a result of repeated radiographic examinations following skull fracture. During the last two decades 7 patients with verified leptomeningeal cysts have been studied at this institution.

CASE REPORTS

Case 1. Unit No. N.I. 9400. M.K., an 18-year-old girl, was admitted on July 2, 1929, complaining of right-sided convulsions and a left parietal skull defect which had been first noticed in childhood and had enlarged progressively. At the age of 16 months she had been struck by a train. The details of the accident and the period of unconsciousness are not known.

Neurological findings on admission were a left parieto-occipital skull defect and slight awkwardness of the right hand. Roentgenograms of the skull revealed an irregular skull defect, 8 cm. in length, in the left posterior parietal region.

At operation the dura mater was found to be thinned out about the periphery of the defect and absent in its center. The area was filled with a 3×2×4 cm. cyst which was not in communication with the ventricles. The dural defect was closed with a Cargile membrane.

The convulsions persisted for the 1st postoperative year and then disappeared. The patient has been symptom-free without medication for the intervening 19 years.

Case 2. Unit No. 18791. I.M., a 9-year-old boy, was admitted on Jan. 30, 1934, with the chief complaint of left-sided convulsive attacks since 1933. In 1925, when he was 3 months of age, he had fallen from his carriage and had sustained a skull fracture followed immediately by paralysis of the left arm and leg. The hemiplegia cleared up slowly, but not completely.

He was a well-nourished and well-developed child. Irregular bulging was noted in the right posterior parietal region (Fig. 1 A). There was mild spastic left hemiparesis with slight left hemiatrophy. He was grossly retarded mentally. Roentgenograms of the skull revealed two large defects in the posterior portion of the right parietal bone with scalloping of the margins of the defect consistent with posttraumatic leptomeningeal cyst (Fig. 1 B). Pneumoencephalography showed moderate dilatation of the posterior portion of the body of the right lateral ventricle.

FIG. 1. *Case 2.* (A) Note irregular bulging in right posterior parietal region. (B) Photograph of the bone specimen, showing scalloped erosion at sites of the original fracture and irregular erosion of the inner table produced by the multiloculated cyst.

At operation a multicystic space was found. The dura mater was absent over these cysts, which were partially formed by degenerated brain tissue. The dural defect could not be closed at this time because of uncontrollable bleeding which delayed the procedure, but it was tightly closed at a second-stage operation.

Following surgical intervention the convulsions became localized to the left leg and later disappeared completely; the hemiparesis improved. The mental retardation remained unchanged over an 8-year follow-up period.

Case 3. Unit No. 803803. J.C., a 7-year-old boy, 8 months prior to admission had been struck by a car and rendered unconscious for 3 days. On regaining consciousness he had shown a left hemiparesis which cleared over a period of several weeks.

Neurological findings on admission were left-sided hyperreflexia and left Babinski sign. Roentgenograms of the skull made 4 months after injury were interpreted as showing erosion of bone at the site of an old fracture, probably by a post-traumatic leptomeningeal cyst in the right parietal region.

A bone flap turned over this area disclosed several small depressed fractures overlying a dural defect 2×5 cm. in size. The dura mater was adherent to the

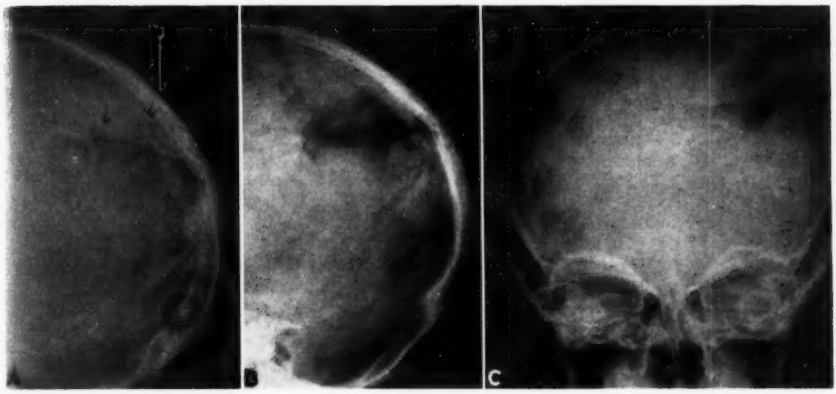

FIG. 2. *Case 4.* (A) Old left parietal fracture showing scalloped margins caused by erosion of the skull from within. (B and C) Lateral and frontal views following injection of oxygen directly into the cyst through the skull defect after withdrawal of fluid. The frontal view shows that the cystic space extends deeply in the skull; the brain was found pushed and flattened (see Fig. 5).

cortex at the margins of this defect and the center of the region was made up of scarred cortex and numerous small multiloculated cysts. The cysts were opened and the dural defect was repaired with a layer of fibrin film.

The postoperative course was uneventful. The patient is now 13 years old and is normal in every respect except for a mild left hyperreflexia.

Case 4. Unit No. 855062. L.A., a 5-year-old girl, was admitted on May 6, 1947, with the chief complaint of convulsive attacks. At the age of 2½ years the child had fallen three stories, striking her head on a concrete surface. She was not unconscious but became blind for several weeks and some right-sided weakness developed. She recovered her vision and was left with only minimal right-sided clumsiness. She also had occasional unilateral convulsive attacks without loss of consciousness.

On admission she was bright and alert, with only minimal right hyperreflexia. Roentgenograms of the skull were interpreted as showing an area of bone erosion with scalloped margins in the line of an old left parietal fracture (Fig. 2 A). Roentgenograms made after replacement of fluid with oxygen through the skull defect revealed the presence of a multiloculated arachnoidal cyst (Fig. 2 B, C).

At operation these cysts were uncapped, the dural-arachnoidal scar was excised and the wound was closed in a water-tight fashion.

Postoperatively the child did well. She developed normally in every respect and was kept on anticonvulsant therapy for 3 years after operation and then this medication was discontinued. She has been free of convulsions for 18 months without medication.

Case 5. Unit No. 946693. J.B., an 8-month-old infant, was admitted to Babies

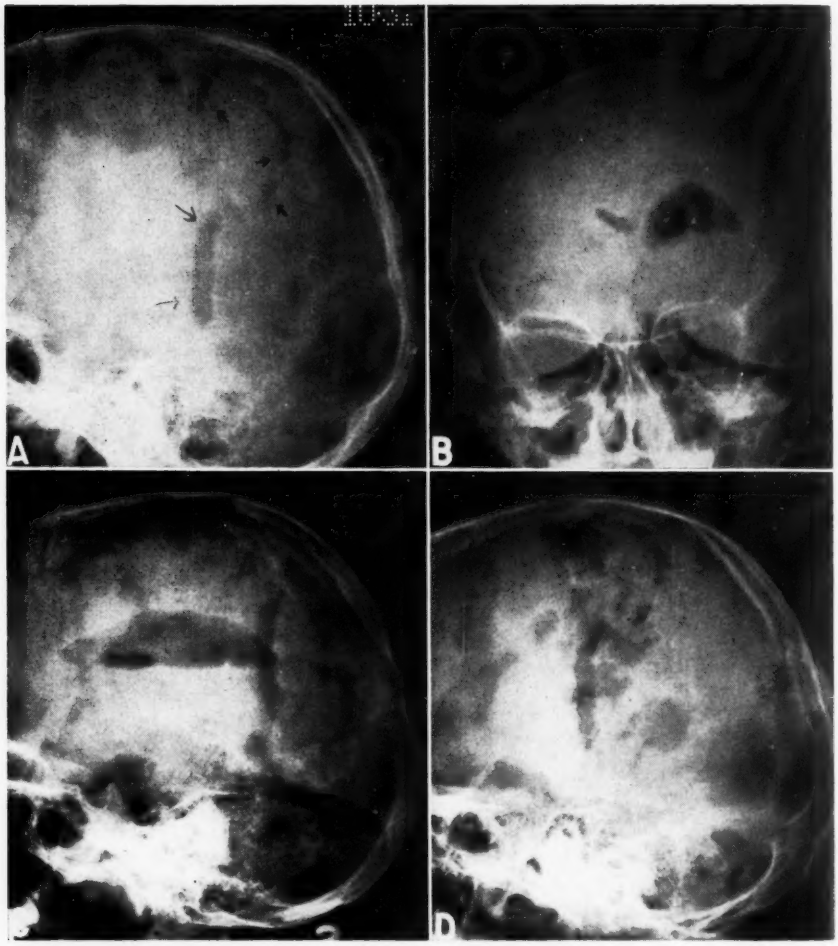

FIG. 3. *Case 6.* (A) One large and several small defects in the parietal bone caused by erosion. (B and C) Frontal and lateral views made in the erect position after pneumoencephalography, showing slight dilatation of left ventricle resulting from post-traumatic atrophy of the brain, a common sequela of severe head injuries. There is no porencephaly or localized dilatation of the lateral ventricles. (D) After exchange of fluid and gas directly through the skull defect 5 days after (B) and (C) a multiloculated cystic space is outlined corresponding with the defects in the plain film; no gas was seen to enter the ventricles or the free subarachnoid space.

Hospital on July 21, 1949 because the parents noticed an enlarging skull defect in the right parietal region.

At the age of 4 months this infant had been dropped from its mother's arms, striking its head on a cement floor. There were no neurological symptoms at the time but a wide linear fracture in the posterior parietal region was found.

Four months later new roentgenograms showed that the fracture site had enlarged to a 7×2 cm. bone defect with sclerotic margins.

At operation a 3×4×3.5 cm. cyst was found under the bone defect. There was no connection between this cyst and the ventricle, and the dura mater was entirely absent over its apex. Several operations were needed in order to achieve a watertight closure of the dura mater; the bone defect was covered with a tantalum plate.

The child has developed normally during the 24-month period of postoperative observation.

Case 6. Unit No. 019241. F.A., an 8-year-old boy, was admitted to Babies Hospital on Oct. 28, 1950, for control of his diabetes of 10 months' duration. The past history revealed that at the age of 15 months he had fallen about 10 feet, striking his head on a stone surface. He had been unconscious for a matter of hours; a severe right hemiparesis was present immediately after the injury and had persisted to a lesser degree up to the time of admission.

Neurological findings were a moderately severe right hemiparesis and a decrease in the circumference of the right limbs.

Plain films of the skull were interpreted as showing an elongated defect, 0.7 by 3.5 cm., and several other smaller defects in the left parietal region (Fig. 3 A). Pneumoencephalography revealed a slightly dilated left lateral ventricle without porencephaly. Five days later, after the gas had been absorbed, oxygen was injected directly through the defect and this revealed a multiloculated leptomeningeal cyst (Fig. 3 B, C, D). No gas was seen to enter the ventricles or the free subarachnoid space at this time.

At operation the bone was found to be eroded in several places and adherent to the underlying dural-arachnoid scar. There was also revealed a 3×4 cm. multiloculated leptomeningeal cyst extending as deep as 2 cm. into the underlying scarred brain substance. This cyst was uncapped, the dural defect was covered with a layer of gelfoam, and the remainder of the wound was closed in the usual manner.

In the 12 months since operation there appears to have been some improvement in the hemiparesis. This is difficult to interpret, however, as the patient has been receiving physiotherapy.

Case 7. Unit No. 068950. P.C., a 4½-year-old girl, was admitted on Nov. 10, 1951, because of severe left hemiparesis and bulging in the right parietal region which had become progressively more prominent. At the age of 2 she had fallen 15 feet, striking her head on a stone walk. She had been semicomatose for about 6 weeks.

Neurological findings on admission were a spastic left hemiparesis with hypoplasia of the left extremities, and a 6×8 cm., slightly bulging skull defect in the right parietal region. Roentgenograms made at the time of the injury were reviewed as were several subsequent films. These series of films demonstrated several fractures of the skull which had united spontaneously while one in the right parietal region had undergone progressive enlargement until it formed a huge defect (Fig. 4, A, B, C). Pneumoencephalography revealed porencephaly under the skull defect.

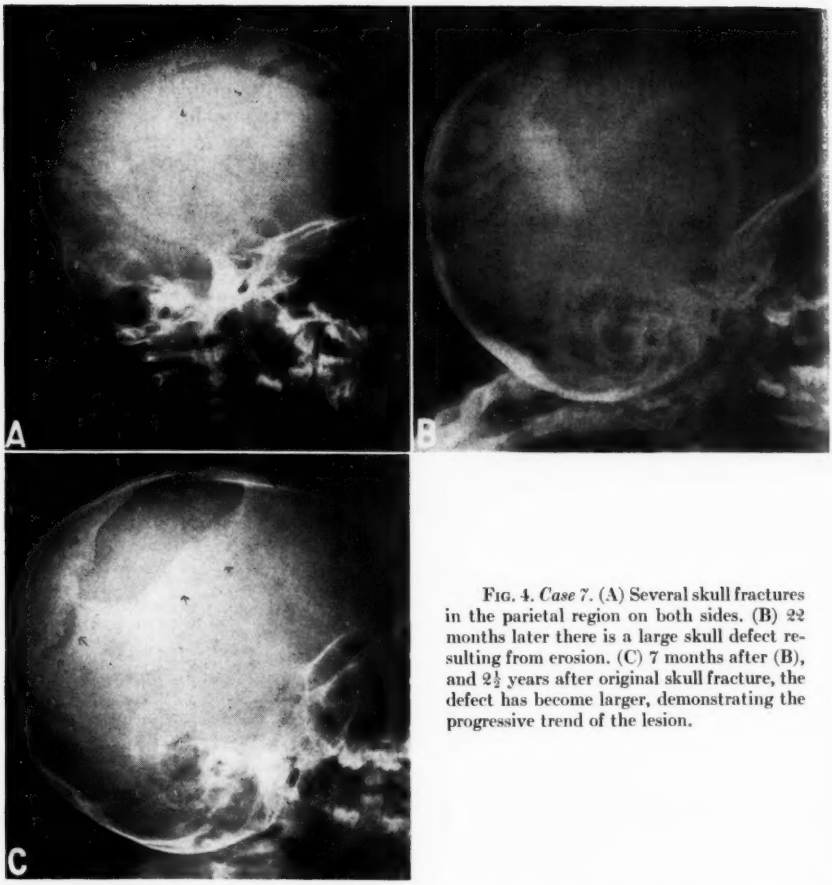

FIG. 4. *Case 7.* (A) Several skull fractures in the parietal region on both sides. (B) 22 months later there is a large skull defect resulting from erosion. (C) 7 months after (B), and 2½ years after original skull fracture, the defect has become larger, demonstrating the progressive trend of the lesion.

At operation shredded-appearing dura mater was reflected to reveal a large area of multiloculated cysts. These were uncapped and the entire area was connected surgically to an underlying dilated ventricle through a layer of scarred white matter 2 to 2½ cm. thick. The dura mater was repaired with Cargile membrane and the bone defect was covered with a tantalum plate.

The postoperative course was complicated by a collection of cerebrospinal fluid under the skin flap necessitating a reclosure of the dural defect. The patient has not been followed long enough to evaluate her course.

ANALYSIS OF CASES

Time of Injury and Development of Bone Erosion. The trauma occurred in childhood. The oldest patient of this series at the time of injury was 5 years and the average age was 2 years and 7 months. The time elapsed

between the injury and the discovery of bone erosion by x-ray examination varied from 4 months to 14 years, but in most cases it was less than 3 years.

Type of Injury. All patients had received a sharp blow to the head having the mechanical characteristics necessary to produce a skull fracture. Five of the children had been dropped or had fallen on their heads on cement pavements for distances varying from 4 to 30 feet. The other 2 children had been struck by rapidly moving vehicles.

Severity of Brain Damage and Leptomeningeal Cyst Formation. No relationship could be established between the development of the cyst and the severity of the brain damage. Two of the patients had rather severe neurological deficit (spastic hemiplegia and hemiatrophy) and 3 had had preoperative seizures. In 1 patient there was gross mental retardation.

Radiologic Findings. The roentgenograms in this condition are so characteristic that a correct diagnosis usually has been made on the basis of plain films. The changes were originally described by Dyke[1] and by Schwartz.[2,3]

The films disclose an irregular defect in the bone, usually with scalloped margins and some degree of sclerosis of the adjacent bone; both inner and outer tables are involved, the inner table to a greater extent. In some instances the old fracture line may be clearly seen at the ends of the skull defect. In all of the 7 cases the lesion is situated in the parietal region and in 6 of them in the posterior half of the parietal bone close to the superior portion (Fig. 2 A, 3 A).

A preoperative diagnosis can be established by injecting oxygen or air through the skull defect after the withdrawal of fluid from the cyst. In the cases in which this was done a multiloculated irregular cystic space was shown and the injected gas did not enter the free subarachnoid space or the ventricles (Fig. 2 B, C and 3 D).

Pneumoencephalography was performed in 3 cases: In 2 (Cases 2 and 6) the lateral ventricle was moderately dilated on the side of the skull defect without evidence of porencephaly (Fig. 3 B and C). In addition to the cyst the other patient (Case 7) had a dilated ventricle with porencephaly. The question may be raised as to the possible role of the porencephalic dilatation in the production of the bone defect, but porencephaly alone rarely causes pressure erosion in the skull.

In the 2 cases in which we have been able to obtain the films made at the time of the initial injury, the original fracture is wide, measuring as much as 4 mm. in width. On the basis of these 2 observations no definite conclusions can be reached, but they suggest that perhaps in every case in which a fracture appears wide when first seen, the patient should be followed for several years, or until the fracture heals, since it would appear that the wider the original fracture the greater the possibility of dural tear and arachnoid herniation with subsequent formation of leptomeningeal cysts. This applies more specifically to children.

Roentgenologically the differential diagnosis of the condition is relatively

easy: With a clearcut history of skull injury several months or years before, the presence of an elongated skull defect with scalloped margins and irregular sclerosis of the bone involving both tables of the skull and usually situated in the posterior parietal region is almost pathognomonic. The skull defects sometimes seen during healing of a cephalohematoma should not cause confusion. With a leptomeningeal cyst there is no evidence of a shell of subperiosteal new bone as is seen in cephalohematoma.

Operative Findings. In all cases at operation the dura mater was found to be absent in the center of the bone defect and markedly thinned and adherent to the underlying arachnoid about the margins of the lesion. The fluid within the leptomeningeal cyst was a clear colorless liquid grossly resembling cerebrospinal fluid. In one case in which the cyst fluid was analyzed chemically it was found to be identical with cerebrospinal fluid. An active search was made for a communication of the cyst with the underlying ventricle, but none was found in any instance at operation.

DISCUSSION

It is our opinion, based on the factors mentioned above, that the mechanism involved in the production of this syndrome is as follows (Fig. 5):

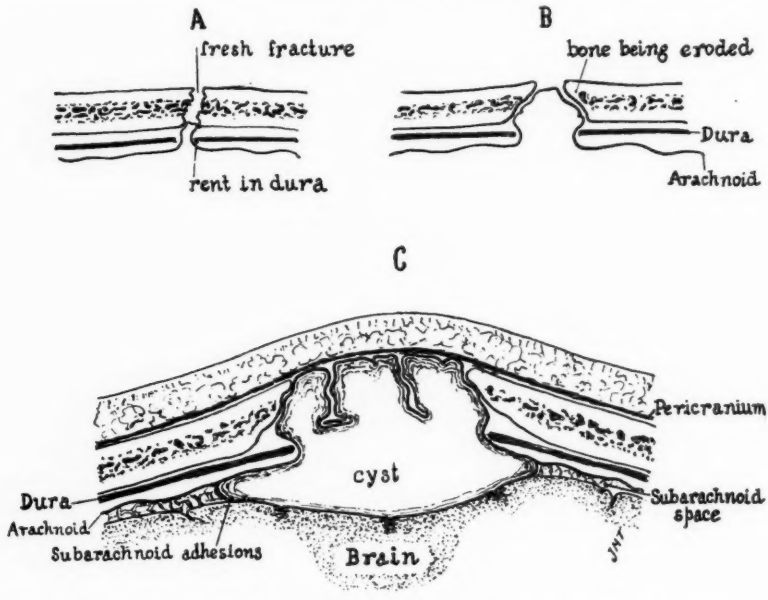

FIG. 5. Diagram showing our conception of the mechanism of production and the progressive changes leading to the formation of a leptomeningeal cyst and erosion of the skull. (A) The original fracture with tear of the dura mater and herniation of the arachnoid membrane. (B) Widening of the dural aperture and beginning erosion of the skull. (C) The final stage when there is marked erosion of bone from within, with further widening of the dural aperture, arachnoidal adhesions around the margins, and depression of the brain.

Trauma produces a skull fracture and an underlying dural tear. At the same time there is probably sufficient subarachnoid hemorrhage to hinder the local circulation of cerebrospinal fluid. The arachnoid membrane projects out through the dural tear into the fracture site. This trapped arachnoidal hernia, aided by the normal pulsations of the brain, gradually erodes the edges of the bone and at the same time compresses the underlying cortex. There must be some degree of a ball-valve mechanism at work also, with the cerebrospinal fluid having easier ingress into than egress from the cyst. Arachnoidal adhesions about the margin of the lesion probably also play a part in trapping the fluid locally. It seems probable that the dural tear is the single most important factor in the pathogenesis of these lesions and that without it the fractures would heal as expected (and as indeed other skull fractures healed in these same patients).

Treatment of these lesions consists of excision of the leptomeningeal cyst, repair of the dural aperture and plating the resulting bone defect if necessary. Since this condition is progressive, early diagnosis is important if one is to avoid extensive erosion of bone and dura. The possibility of minimizing the underlying brain damage and lessening the severity of post-traumatic epileptic attacks also constitutes good reason for early therapy. Two of the 3 patients with preoperative convulsions became seizure-free following surgical repair of the lesion. Because diagnosis is the first step in therapy, we feel that roentgen examination of the skull at 3 and 6 months following injury is indicated in children who have sustained skull fracture, particularly if the fracture is in the parietal region.

SUMMARY

Seven cases of post-traumatic cerebral leptomeningeal cysts are reported. The bone along the fracture was eroded and the fracture defect was irregularly widened.

Analysis of the factors common to all these cases indicates that rupture of the dura mater and herniation of the arachnoid into the fracture is probably the mechanism by which the lesion is formed.

Because of the importance of early diagnosis, it is advisable to have repeated x-ray examinations of the skull following fracture of the skull in children at intervals of 3 months until the fracture heals.

We would like to thank very specially Dr. E. H. Wood and Dr. W. Brigham for their valuable help in the preparation of this paper.

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COLLOID CYSTS OF THE THIRD VENTRICLE

REPORT OF SEVEN CASES

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OF THE true intraventricular tumors, colloid cysts of the 3rd ventricle form an interesting and favorable group. Previous to the advent of cerebral pneumography, reported cases were mere pathologic curiosities. Their clinical diagnosis and surgical removal were made possible by the pioneering work of Dandy[6] who, in 1933, reported 5 cases with successful results in 4. A number of reviews have since appeared in the literature.[16,26,29,33] McLean[16] in 1936 and Zeitlin and Lichtenstein[31] in 1937 particularly discussed the pathogenesis. In a later publication, in June, 1940, the latter authors[32] stated that there were 68 known cases to that date. About 22 cases have been added to the literature since then.[1,2,4,11,18,19,24,25,27,29,30] As experience accumulated, more and more of the reports described the successful surgical removal of these cysts. Although rare, colloid cysts of the 3rd ventricle are remarkably similar pathologically and while vagaries in the clinical picture occur, the radiographic and operative aspects in the management are readily standardized. As these are favorable tumors, permanent and often dramatic results have been attained after extirpation. On the other hand, certain undesirable sequelae may occur in patients from whom such a cyst has been removed successfully by the usual transventricular procedure. It is for this reason that further information regarding the eventual history of these patients is needed in order that operative as well as other data may be evaluated.

The purpose of this paper is to record the experiences of the members of the neurosurgical department of the Lahey Clinic with 7 patients who had cysts of the type mentioned. That they are rare lesions is shown by the fact that they represent only 0.55 per cent of the total of 1,256 patients who had surgically verified brain tumors treated at the clinic during the period from 1934 through 1945.

REPORT OF CASES

Case 1. A 33-year-old white man was admitted on July 23, 1934. For "many years" he had had severe headaches across the vertex usually once a week. Recently, the headaches had been in the occipital region and extended forward to the frontal region. They occurred two or three times a week, were precipitated by emotional upsets and were accompanied by nausea and nonprojectile vomiting. For the last

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6 months he had complained of frequent "dizzy spells" and increased unsteadiness n walking. Changes in vision had not been noticed. The past history was noncontributory except for venereal exposure 10 years previously. A blood Wassermann est in 1927 and a Kahn test in 1934 were negative. He was said to have had a peptic ulcer some years ago which was cured by diet.

Examination. The patient was somewhat thin and spare in build. He was mentally clear and cooperative. The eyes were slightly prominent. The right pupil was larger than the left; both were regular and reacted normally to light and in accommodation. The extra-ocular movements were normal; there was some fine nystagmus in both lateral directions and a fairly coarse nystagmus was present in the upward gaze. The fundi showed obliterated disk outlines with papilledema of 3 to 4 D. Marked venous engorgement and tortuosity were present and considerable new tissue formation was noted, especially over the left disk. There was an equivocal Babinski sign on the right. Finger-to-nose and finger-to-thumb tests were done fairly well but there was a distinct tremor on the right side in the finger-to-nose test. There was pronounced adiadochokinesia on the right on sustained testing. The heel-to-knee test revealed some dysmetria and a coarse tremor on both sides, more marked on the right. Blood Hinton test was normal.

Roentgenograms of the skull showed a thin vault with fairly definite signs of increased pressure. The sella turcica was atrophic and the posterior clinoids appeared pushed back without erosion of the dorsum sellae.

On July 24, 1934, ventriculography showed enormously dilated lateral ventricles with no filling of the 3rd ventricle (Fig. 1).

1st Operation. Because of the fairly marked clinical evidence of incoordination.

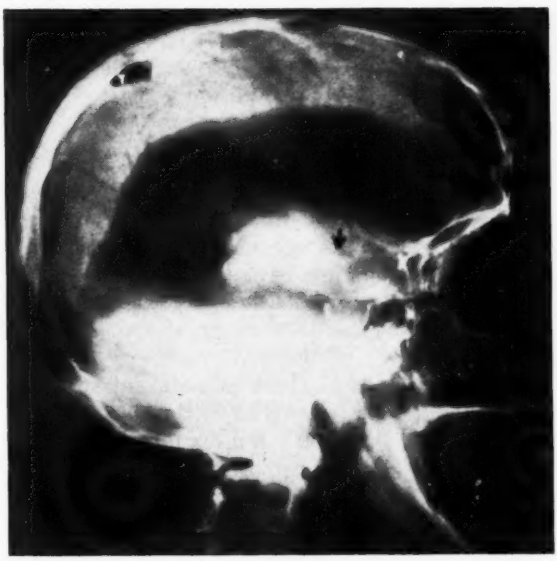


FIG. 1. *Case 1.* Ventriculogram showing greatly dilated lateral ventricles, with a broad foramen of Monro and no filling of the 3rd ventricle. The posterior clinoids lean slightly backward.

it was thought that the patient had a cerebellar tumor; therefore, a suboccipital exploration was performed (by G.H.) through a cross-bow incision. There was a large posterior cistern with a gray, thickened wall, but no evidence of tumor in the posterior fossa was disclosed.

2nd Operation. On July 26, 1934, under local anesthesia, a moderate-sized anterior bone flap was turned down on the right side. Through an oblique 4 cm. incision made in the cortex into the right lateral ventricle a cystic tumor filled with mucoid gelatinous material was removed.

Course. The postoperative period was characterized by fluctuating states of consciousness which varied between voluntary speech and ability to recognize relatives to deep coma. Frequent ventricular taps were required. On the 3rd postoperative day, a ventricular needle was left *in situ* for varying periods of time to institute constant drainage of the ventricles. This procedure improved the patient's responsiveness and reduced the pressure at the site of the decompression for 4 weeks. A left facial weakness and an external deviation of the eye developed; the latter disappeared on the 8th postoperative day when the patient was in relatively good condition, but it reappeared after the 18th day. Rigidity in flexion and tremor of the right arm persisted after the 22nd day. Lumbar punctures did not relieve the pressure at the site of the decompression. His fever varied from 101° to 103°F. during the periods of coma, but hyperthermia was not noted otherwise. It was evident that a normal CSF circulation would not be established without further surgical intervention.

3rd Operation. On Aug. 28, 1934, 33 days after removal of the cyst, the osteoplastic flap was again elevated and the old incision in the cortex was reopened. The septum pellucidum was adherent to the lateral wall of the right lateral ventricle in such a way as to occlude the foramen of Monro when the ventricles were filled with fluid. The 3rd ventricle was filled with old blood clots.

Course. After this procedure, the patient steadily deteriorated. Temperature, and pulse and respiratory rates rose progressively. His temperature was 108.5°F. at the time of death, 1 day after operation. Permission for autopsy was not obtained.

Comment. The preoperative diagnosis of a posterior fossa tumor was based on the history of staggering and the clinical evidence of incoordination. It is evident in reviewing the history, however, that his symptoms were intermittent so that he was able to continue working and the signs that pointed to a posterior fossa lesion were inconstant. Nevertheless, at the time the ventriculograms were first read, it was believed that the lesion was posterior in spite of the fact that filling of the 3rd ventricle was not evident.

Two other cases are recorded in which suboccipital explorations had been done with negative findings. In Case 3 of Fulton and Bailey[10] the patient survived the operation, but died after a lumbar puncture was done 2 months later. In Case 2 of Dandy's[6] series exploratory operations were done for 3 years before a diagnosis of colloid cyst was made.

At the third operation in our Case 1, adhesion of the septum pellucidum and the old blood clot in the 3rd ventricle prevented the normal egress of the ventricular fluid from the lateral ventricles. In the case of Campbell and Whitfield[3] adhesions which closed the interventricular foramina were

corrected successfully at reoperation. We have since included the making of a large opening in the septum pellucidum as part of the operative procedure, as recommended by Weinberger and Boshes.[29] This insures free communication between the two ventricles and continuous cerebrospinal fluid circulation and absorption in the event that one foramen of Monro should become occluded after operation.

Case 2. A 42-year-old man, a bank clerk, was admitted on June 19, 1935. Five weeks previously he had been marching in a parade and had been forced to drop out of line because of sudden intense headache, diarrhea and vomiting. He had returned to work for a few days, but was unable to continue thereafter because of paroxysmal headaches referred to the right eye. Ten days before admission he had a sudden, severe attack of vomiting which lasted approximately 6 hours. His wife stated that for some time before the onset of his headaches, difficulty in orientation as to time, and changes in mentality and habits had been present. In the past year, there had been a slight diminution in libido. Several headaches were associated with nausea and vomiting, blurring of vision, and disturbances in the right visual field in the form of waves without particular shape. Recently he had a transient "numbness and prickling" of his left hand in the metacarpophalangeal area of the 3rd and 5th fingers. Five days before admission, he was examined by a physician who found that the patient was disinterested in the discussion of his condition, exhibited lack of attention and showed a tendency to sudden bursts of silly laughter. Ophthalmoscopic examination revealed early choking of the disks. Three days later, an increase in the degree of the papilledema was noted.

Examination. The patient was fairly alert but was unable to concentrate. He showed a tendency to repeat every question and apparently had no insight into his condition. The pupils were widely dilated but reacted normally to light and in accommodation. The fundi showed complete obliteration of the disk margins with an elevation of 2 D. Although accurate visual fields could not be obtained there was a suggestion of a left upper partial homonymous quadrantic defect and a general constriction of the fields. The external ocular movements were normal. There was slight left lower facial emotional weakness. The deep tendon reflexes were exaggerated on both sides, although the responses were quicker on the left. Babinski sign was positive on the left. The heel-to-shin test was done jerkily on both sides, although the impression was that there was no true ataxia.

Roentgenograms of the skull showed marked convolutional atrophy throughout the frontal and parietal regions on both sides. The sella turcica measured 13 mm. in length and 10 mm. in width; the floor was smooth and even in contour. The dorsum sellae was normal. The anterior clinoids showed distinct decalcification on both sides.

Operation. Ventriculography and craniotomy (by G.H. and J.L.P.) were done on June 24, 1935. There was marked symmetrical dilatation of both lateral ventricles, without distortion or displacement. The 3rd ventricle was only partially filled. Its midportion was occupied by a small, rounded mass, about 1.5 cm. in diameter, located between the interventricular foramina, which were partially visualized.

A right frontal bone flap was turned down and a vertical transcortical incision was made in the frontal lobe well anterior to the motor area (Fig. 2). This was carried down into a widely dilated ventricle. The interventricular foramen was enlarged anteriorly and below it the wall of a yellowish-gray tumor was exposed. The septum

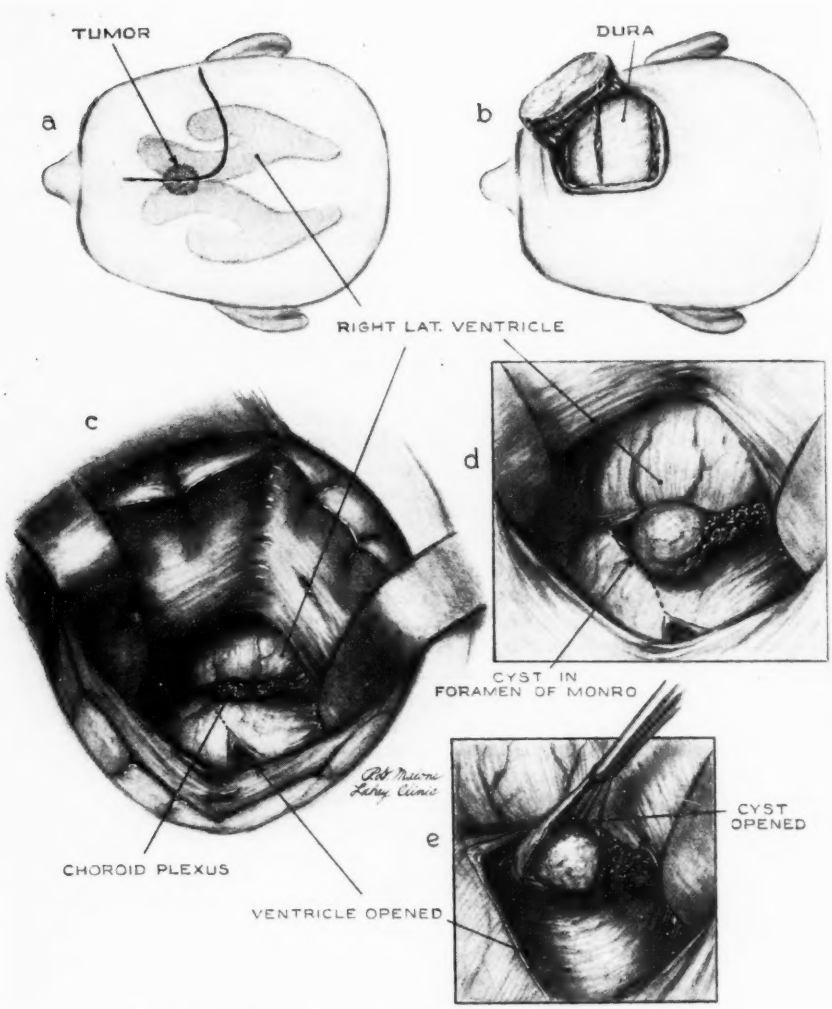


FIG. 2. *Case 2.* Drawings at surgery, showing the operative approach and removal of a colloid cyst of the 3rd ventricle.

pellucidum was then incised from the margin of the foramen upward for a distance of approximately 3 cm., where a septal perforation was present. A somewhat irregularly shaped cyst, 1.5 to 2 cm. in diameter, was disclosed in the anterior 3rd ventricle. A small electrosurgical incision was made in the cyst and 6 cc. of sticky mucoid material were evacuated. The cyst wall could then readily be removed.

Course. A left hemiplegia and a left facial weakness developed immediately

after the operation, but markedly improved thereafter. For 1 week the patient was unresponsive although apparently he was aware of his surroundings, and he was lucid at intervals. Two weeks after operation a neurologic review revealed that he cooperated well, but was rather facetious, with a tendency to elaborate verbal responses. He had normal olfaction on the left and demonstrable hyposmia on the right. Visual function was normal on gross tests. There was regressive papilledema with an elevation of 1.5 D.; the right disk showed slight secondary optic atrophy. Pupils were equal and reacted to light and in accommodation. There was bilateral 6th nerve weakness, equal on both sides. An upper motor neuron weakness of the left side of the face was present, more marked emotionally than volitionally. The left limbs were slightly weaker and more flaccid than those on the right. There was less brisk response to pinprick and cotton wool on the left than on the right. Heel-to-knee test was done poorly on the left. Quick-succession movements were moderately awkward on the left. Posture maintenance showed a drift of the left hand. The impression obtained was that of left residual weakness of the upper motor neurons. The patient was apparently improving mentally and neurologically at the time of his discharge, 17 days after operation.

By Sept. 17, 1935, 3 months after operation, he had made progressive recovery in all respects. He had no complaints of headache or vomiting. Recollection of his hospitalization was extremely hazy. His memory for names was very poor and he was constantly afraid that he would do or say the wrong thing. Physically, he was in excellent condition and had gained 25 pounds. The optic disk outlines were clear except for a slight haziness of the nasal border on the left.

He returned April 1, 1936, 10 months after operation. Five months after operation, an episode had occurred during which he "turned around three times and fell down." He regained consciousness in a hospital, where he stayed for about 5 days; a lumbar puncture was reported to have shown no increase in pressure. The patient did not complain of headache or of difficulty with his extremities. There was slight blurring of the nasal margin of the disks.

Subsequent reports have been received from his wife. Three years after operation, there was residual impairment of memory and ability to concentrate, but otherwise he was well.

Comment. The preoperative diagnosis in Case 2 was right frontotemporal tumor. The positive findings of pyramidal involvement and equivocal visual homonymous field defect were the only localizing signs. The short duration of symptoms suggested a rapidly growing lesion.

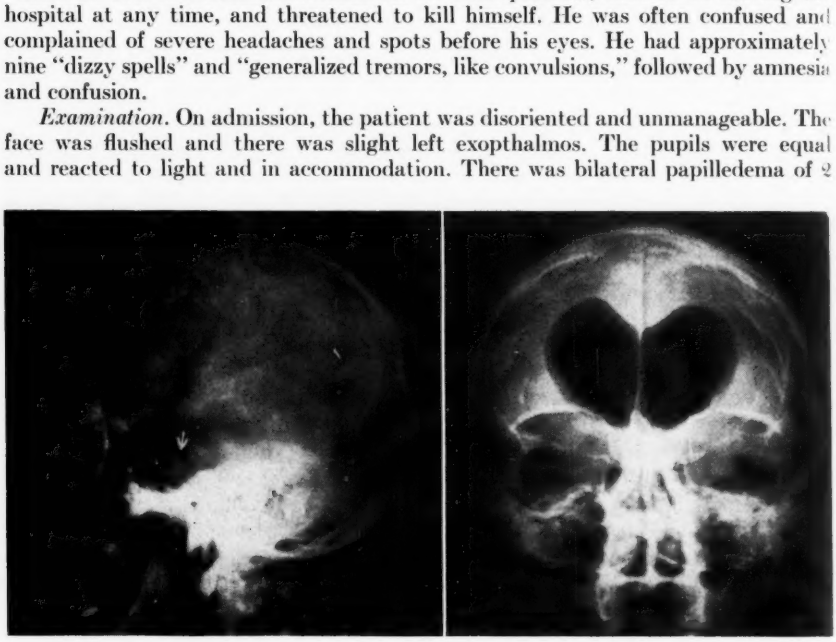
This case illustrates how a colloid cyst of the 3rd ventricle may cause some degree of increased intracranial pressure as evidenced by the fairly advanced cranial changes demonstrated by radiography without, however, producing headache or vomiting in the meantime. In view of the radiographic evidence of long-standing intracranial hypertension, the diminution in libido approximately a year before admission was probably significant and might well be considered to have been the first symptom.

Case 3. A 40-year-old white man was referred to the Lahey Clinic on March 15, 1937. He was unable to give any history of his illness. He had been confined in a state mental hospital since Jan 28, 1937. A sister was confined in a mental hospital. The patient had had severe, intermittent, generalized headaches for 6 years.

They occurred once or twice a month and often had forced him to stop his work in the middle of the day. A year before admission, his appetite was said to have been enormous, to the extent of his consuming two to three times the amount of a normal meal as often as eight times a day. Six months before admission he had had "recurrent amnesia and vertigo."

In the state hospital, the patient had showed poor cooperation and refused to remain in bed. He committed nuisances on other patients, insisted on leaving the hospital at any time, and threatened to kill himself. He was often confused and complained of severe headaches and spots before his eyes. He had approximately nine "dizzy spells" and "generalized tremors, like convulsions," followed by amnesia and confusion.

Examination. On admission, the patient was disoriented and unmanageable. The face was flushed and there was slight left exopthalmos. The pupils were equal and reacted to light and in accommodation. There was bilateral papilledema of 2



FIGS. 3 and 4. *Case 3.* (*Left*) Lateral view of skull showing erosion and flattening of posterior clinoids. (*Right*) Anteroposterior ventriculogram showing internal hydrocephalus without filling of 3rd ventricle.

to 3 D., and secondary optic atrophy, more marked on the right side. There was weakness of the right 6th nerve and slight limitation in the upward gaze. Sustained nystagmus was present in both lateral and upward directions of gaze. There was an emotional left lower facial weakness. There was unsustained bilateral ankle clonus and a questionable Babinski's sign on the left side at one time. He tended to fall to the left each time the Romberg test was performed. The finger-to-nose test showed ataxia on the left side; heel-to-knee test was done slowly and poorly on both sides.

Roentgenograms of the skull showed considerable convolutional markings of the vault and marked atrophy of the posterior clinoids; the latter were upright (Fig. 3). The sella turcica was slightly enlarged.

Operation. On March 18, 1937, ventriculography and craniotomy were done (by G.H.). There was marked dilation of the lateral ventricles without filling of the

3rd ventricle (Fig. 4). A right anterior osteoplastic bone flap was turned down. A transcortical incision was made in the premotor area. A thin-walled cyst was removed through the enlarged lateral ventricle. The patient withstood the procedure very well and no alterations in pulse or blood pressure were noted.

Course. A left hemiplegia developed which gradually improved during the following week. Six days after operation, the patient had a convulsion which involved the left side of the face and the left upper and lower extremities. Daily ventricular taps revealed a persistent xanthochromia of the ventricular fluid; consequently, 15 days after operation, the bone flap was re-elevated as it was thought that possibly there was an intraventricular clot. No evidence of a clot was found although there was considerable edema in the region from which the tumor had been removed. The postoperative course was uneventful thereafter. The hemiparesis cleared and, although he was only mildly interested in his surroundings, he was gradually able to get up and his behavior improved. He had no further convulsions. He became amenable to suggestion and did not interfere with other patients as he had done before operation. The optic disks showed marked recession of the papilledema and his neurologic status was satisfactory at the time of discharge, 40 days after operation.

The patient was readmitted to the state hospital and following is a summary of periodic hospital reports.

June 17, 1937. All symptoms had disappeared. There were no headaches, convulsions, or vomiting spells and his general health had been good. Mentally, he was completely out of touch with his surroundings, disoriented in all spheres and did not remember persons, places or his way around the hospital. It was noted that he ate normally and had lost his unusual craving for food which had been observed during his previous hospitalization.

May 4, 1938. He had been free from headache and dizziness except on a few occasions. He had a convulsion 11 months after operation. His appetite was normal and his physical condition was good.

Feb. 2, 1939. He had been completely free from headaches. In the past year, he had had spells during which he tore bedding and clothing and became excessively angry and uncontrollable when he was accused of doing so. Neurologic findings showed no changes; eyegrounds were normal.

Sept. 14, 1940. There had been little change in the past $1\frac{1}{2}$ years except for occasional convulsions. These were severe and were followed by confusion for as long as 72 hours. Not infrequently after an attack, he had a fever of 103°F. for a period of time. At times he was quiet and agreeable; sometimes he was irritable, quarrelsome and inclined to be noisy. The patient died following two convulsions on Oct. 27, 1941 ($4\frac{1}{2}$ years after operation).

Comment. The preoperative diagnoses in this case were (1) 3rd ventricle tumor, probably colloid cyst, and (2) posterior fossa tumor. The first was considered more likely on the basis of a long clinical history and the radiographic picture of the sella turcica. In the literature on colloid cysts of the 3rd ventricle, several other cases have been reported with predominating clinical signs of mental abnormality. In Case 1 of Riddoch[23] a clinical picture of progressive dementia was presented; the cyst was diagnosed by ventriculography, but the patient died without operation. Larson's[15] case was

diagnosed as psychosis with arteriosclerosis and the patient was not given surgical treatment. It was complicated by evidence of rupture of the cyst into brain substance. Of Jefferson and Jackson's[13] cases, one patient had recurrent psychotic episodes of delusion and another had marked mental disturbance. In Neustaedter and Meltzer's[20] case the patient was committed to a hospital for the insane because of marked behavior changes. Perusal of other case reports also shows a high incidence of objective mental changes, particularly in those in whom there was evidence of long-standing hydrocephalus. It is evident in our case that the brain had already sustained considerable damage before treatment was instituted and, although the patient survived for 4½ years following operation and was without a recurrence of headaches, he was permanently disabled mentally. A familial predisposition should, however, be considered as having possibly contributed to a certain extent to the final picture.

Postoperative edema in the region of the 3rd ventricle, as was found in this case, may prevent establishment of a normal flow of ventricular fluid during the immediate postoperative period and an intraventricular clot could be justifiably suspected. Unless definite signs of hemorrhage become evident or deterioration in the condition of the patient supervenes, it is perhaps best to maintain the patient by means of ventricular punctures or continuous ventricular drainage before an exploratory operation for a clot is undertaken.

Case 4. A 20-year-old woman bookkeeper was admitted on May 1, 1939. She had been well until 2 years previously when she first began to have headaches which were associated with vomiting and pain in the back of the neck. At the onset, the headaches had occurred about once a week, but at the time of admission they were continuous. They were chiefly suboccipital in location, extended forward, and were brought on by excitement. At times, she noted that with her headaches there was tremor throughout her body. Other accompanying symptoms were nausea and weakness. For the last year she had had attacks of nervousness which were worse during her menstrual periods and described as a "tremble and a feeling of wanting to cry." She believed that she had a staggering gait, with a tendency to drift to the right, but this was not noted by other people. During the last 3 to 4 weeks she had complained of dizziness and blurring of vision which were brought on by sudden rapid movements.

Examination. The patient was an intelligent, cooperative, young woman. There was no anosmia. The pupils were equal and reacted to light and in accommodation. The optic disks showed bilateral papilledema of 4 D., and slight optic atrophy. Her visual acuity was slightly impaired and the gross visual fields were full. The external ocular movements were normal except for some nystagmoid jerks on lateral directions of gaze.

Roentgenograms of the skull showed a vault of average thickness with diastasis of the sutures. The sella turcica was slightly enlarged, with depression of the floor in the midline and squaring of the superior margin of the posterior clinoids, suggestive of a tumor above the sella.

Operation. Ventriculography was done May 3, 1939. There was marked dilatation of the lateral ventricles without displacement or deformity. There was no air in the

3rd and 4th ventricles. Diagnosis: Third ventricle tumor (Fig. 5). A right frontal craniotomy was then done (by J.L.P.). A transcortical incision was made through the anterior portion of the frontal lobe into the anterior horn of the right lateral ventricle. A thin-walled bluish cyst, 1.5 cm. in diameter, lying in the anterior portion of the 3rd ventricle, was removed.

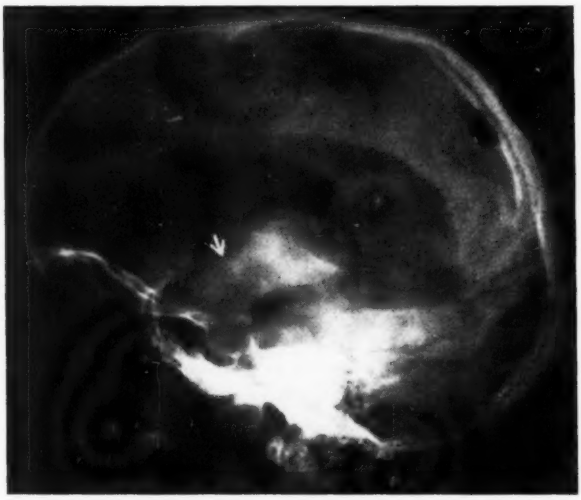


FIG. 5. *Case 4.* Ventriculogram showing great dilatation of the lateral ventricles without filling of the 3rd and 4th ventricles; no changes in the sella turcica except squaring off of the posterior clinoids.

Course. The postoperative course was somewhat difficult and was marked by rises in temperature. Daily lumbar punctures were done for 8 days and every other day thereafter for 10 days. The patient then became afebrile, improved rapidly and was discharged on the 25th postoperative day. Her only complaint was that of impaired memory. Objectively, this was manifested by inability to recall events that had occurred 24 hours previously.

She was completely free from symptoms for several months after discharge. A seizure which involved the left upper and lower extremities occurred 10 months after operation. This was preceded by turning to the left and was followed by transient postictal left hemiparesis. At examination 2 days later, residual findings were not noted and the optic disks were normal.

The patient passed an examination for life insurance 3½ years after her operation and was married 6 months later. In a letter written in December 1950 (11½ years after operation) she reported that she worked daily in addition to managing a home. Her last convulsion occurred in 1943.

Comment. The preoperative diagnoses in this case included: (1) brain tumor, unlocalized (possible 3rd ventricle tumor or frontal tumor) and (2) possible cerebellar astrocytoma. Increased intracranial pressure was evident clinically and in the roentgenograms; however, there was a paucity of local-

izing signs. Increased intracranial pressure without tumor was not considered likely because of the long duration of the headaches. The only positive neurologic findings were the choked disks and moderate hypotonia. The ventriculograms were diagnostic and the operation was reasonably successful.

Case 5. A 42-year-old tax collector was admitted on Feb. 25, 1940. For 11 years he had had headaches at irregular intervals. They were referred to the right side of the vertex and to the occipital region, and were described as a feeling that "the top of his head was coming off." The headaches lasted from 5 to 10 minutes, came suddenly and just as suddenly receded. Occasionally, they were relieved by work. His teeth were removed because they were said to have been very brittle; they were not abscessed at the time.

Although subject to headaches, he remained reasonably well until 6 months before admission when he began to have momentary losses of equilibrium, which he noted as a tendency to veer to the left while walking. Two months later he complained of sudden, frequent weak spells. He had had to lie down during these periods, otherwise he thought that he would collapse. At times, his left leg felt like "pins and needles," appeared to drag when he attempted to walk and was hard to lift. Apparently the spells were absent during periods when he was at work and always occurred in the afternoons. During the last 3 weeks the spells had occurred 2 or 3 times a week. Since a radical mastoidectomy in his childhood, the patient had been deaf in the left ear.

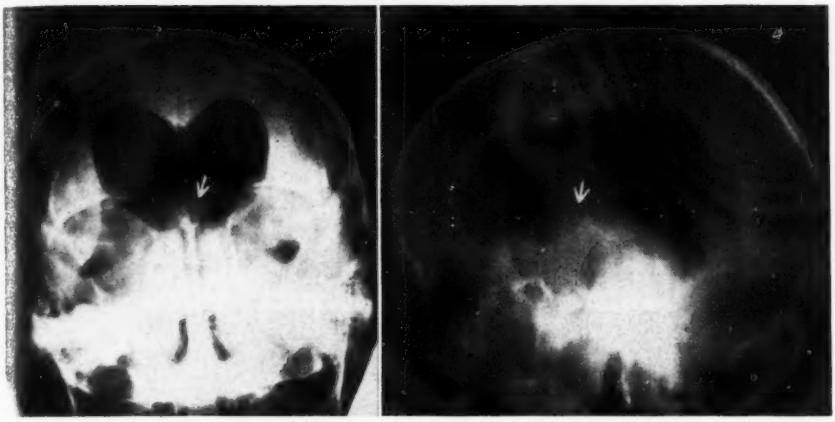
Examination. The patient was well developed and slightly obese. The skin was soft, smooth and almost devoid of hair on the arms and legs. The pubic hair was sparse and feminine in distribution. The pubic pad of fat was somewhat overdeveloped, the penis was small, and the testes were normal in size.

The pupils were equal and reacted to light and in accommodation. External ocular movements were normal. Eyegrounds were normal. There was deafness of the left ear. The left arm and leg were slightly more sensitive to pinprick tests than were the right. He had a memory defect for recent events and exhibited a tension state not unlike that seen in chronic alcoholism.

Roentgenograms of the skull showed a thin cranial vault with definite signs of increased intracranial pressure. The pineal body was displaced to the left and downward. The sella turcica showed erosion and thinning with partial fracture and erosion of the posterior clinoids. The right anterior clinoid appeared thin and elevated. There was slight erosion of the medial edge of the sphenoid wing close to the left anterior clinoid. The findings were interpreted as suggestive of a frontal lobe tumor.

Operation. Ventriculography on Feb. 26, 1940 showed greatly enlarged lateral ventricles, slightly more marked on the right side. The interventricular foramina appeared dilated, each with an apparently abrupt occlusion; air was not seen in the 3rd ventricle. A rounded filling defect protruded between the lateral ventricles in the anteroposterior projection; the shadow was larger on the right side (Fig. 6). This was well demonstrated in the sagittal laminagram (Fig. 7). Diagnosis: Anterior 3rd ventricle tumor.

At operation (by J.L.P.) an osteoplastic bone flap was turned down on the right side. Through a frontal transcortical incision a cystic tumor, 2 cm. in diameter, was removed. The septum pellucidum had been completely absorbed with the exception of some veins that had run through it which looked like guy ropes extending from the corpus callosum to the roof of the 3rd ventricle.



FIGS. 6 and 7. *Case 5.* (*Left*) Ventriculogram showing the tumor shadow protruding in the midline between symmetrically dilated ventricles (arrow). (*Right*) Median sagittal laminagram showing the outline of the tumor shadow (arrow).

Course. There was some degree of disorientation for a few weeks. The patient was mentally clear when he was discharged 32 days after operation.

Ten days later he reported that he had experienced a strange sensation that a black cloud was rolled upward from over his eyes and things became clear to him for the first time since the operation.

Two months and 10 days after operation, neurologic findings were normal. The patient remained completely well and was gainfully employed when last heard from on Dec. 19, 1949, 9 years and 10 months after operation.

Comment. This is the only case in our series in which papilledema was not present. The impression after the first examination was (1) possible neurosis and focal cerebral lesion, and (2) possible hypopituitarism. The plain skull films showed evidence of increased intracranial pressure and localized parasellar changes, suggestive of a frontal lobe tumor. Ventriculography, however, revealed a defect protruding between the lateral ventricles in the region of the interventricular foramina. This was well demonstrated by midline sagittal laminagrams as well as by conventional films (Figs. 6 and 7). The pineal shift to the left is explained by the larger ventricle on the right. This asymmetry was probably caused by unequal degrees in obstruction of the interventricular foramina.

According to McLean,[16] discharge, at times, of the noninspissated cystic content through minute openings in the cyst wall may account for the occasional occurrence of long clinical remissions and eventually for the almost cholesteatomatous character that the content may assume.

Case 6. A 25-year-old white woman was admitted on May 27, 1941. She had been healthy until about 2 years previously when she began to have irregular headaches, almost always associated with nausea and vomiting. They usually occurred at night and persisted for 24 to 36 hours. They were dull in character, began in the frontal region or the occiput and radiated either backward or forward.

She also complained of pulsating noise in both ears, more marked on the left side when she retired. Occasionally, on movement of the head, she noticed dizziness without rotation of objects. Two months prior to admission, an appendectomy had been performed under spinal anesthesia; the appendix was chronically inflamed. Following the operation, severe headaches, with nausea and vomiting, developed.

Examination. The pupils were equal and reacted to light and in accommodation. External ocular movements were normal. There was bilateral, fine, unsustained nystagmus on both lateral directions of gaze. The visual fields grossly were full. The optic disks showed papilledema of 2 D., and a small retinal hemorrhage on the left side. Deep tendon reflexes were active and equal on both sides. Abdominal reflexes were absent on the right and feeble on the left.

FIG. 8. *Case 6.* Median sagittal laminagram showing no filling of the 3rd ventricle, some air in the callosal sulcus, and a broad foramen of Monro.

Roentgenograms of the skull were normal.

EEG on May 29, 1941 showed abnormal, slow activity arising from all areas, most marked in the posterior parietal leads.

Lumbar puncture on May 27, 1941 showed an initial pressure of 270 mm. of water. The fluid was clear and acellular; total protein 30 mg./100 cc. Blood Hinton test and CSF Wassermann, Hinton and gold sol reactions were negative.

Operation. Ventriculography on June 2, 1941 (Fig. 8) showed considerable symmetrical dilation of both lateral ventricles. There was no air in the 3rd and 4th ventricles.

A right frontal craniotomy was performed (by G.H.) on June 4, 1941. The dura mater and brain appeared normal. A small cone of frontal lobe (2 cm. by 1 cm.), about 2 to 2.5 cm. posterior to the tip of the frontal lobe, was removed down to the lateral ventricle. A bluish spherical mass, about 1.5 cm. in diameter, was removed from the anterior 3rd ventricle. A small opening was made in the septum pellucidum before closure.

Course. Beginning on the 5th postoperative day, the patient complained of headache and became drowsy, disoriented, and incontinent of urine at times.

Lumbar puncture on the 7th day showed an initial pressure of 130 mm.; the fluid was clear and pale yellow. On the 8th day, the pressure was 245 mm. and on the 9th day, 130 mm. Repeated punctures relieved her headaches. She continued to improve until the 9th day when, while conversing normally with some friends, she suddenly became cyanotic and died. Permission for autopsy was not obtained. Pulmonary embolism was the probable cause of death.

Case 7. A 21-year-old white graduate nurse was admitted on July 21, 1945. Two years previously she had started to have intermittent occipital headaches accompanied by nausea and vomiting. The headaches were always sudden in onset,

FIG. 9. *Case 7.* Ventriculogram showing partial filling of the 3rd ventricle. Tumor shadow is clearly outlined at the foramen of Monro, which is considerably widened. Posterior clinoids are bent forward and down.

slow in recession and usually lasted 2 to 3 hours. At first they occurred about once a month but recently they had increased in frequency to about once every 2 weeks. Coincident with the onset of headaches, the patient had noted a constant swishing noise in the right ear which was synchronous with her pulse. At about the same time she had transient diplopia and episodes of dizziness which were not associated with vertigo but she had a tendency to fall. One year before admission, she began to experience occasional incoordination in walking associated with a feeling of heaviness of the legs. During the last week her vision had been blurred.

Examination. The patient was a well nourished, well developed woman in no apparent distress. Auscultation of the head revealed a swishing murmur synchronous with the pulse, over the right side of the face and head. The murmur was heard best over the area between the lobe of the ear and the sternomastoid muscle.

There was slight ptosis on the right side. Diplopia was noted on right lateral

gaze. Fine nystagmus was present on both lateral directions of gaze. There was bilateral choking of the disks; 2½ D. on the left and 3½ D. on the right. The vessels were tortuous and there were several fresh hemorrhages around the disks. The visual fields were full to confrontation tests. There was slight hypotonia of the upper extremities, an equivocal Babinski sign on the left side, and the gait showed slight widening of the base.

Roentgenograms of the skull demonstrated that the cranial vault was of average thickness; the convolutional markings were somewhat prominent and the sutures were widened, particularly in the occipital region. The sella turcica was not enlarged and the anterior clinoids were intact but the dorsum sellae and the posterior clinoids were atrophied. The impression obtained from the radiograms was that intracranial pressure was increased.

Operation. Ventriculography on July 24, 1945 showed dilatation of the lateral ventricles, more marked on the right side. The 3rd ventricle was partially visualized and there was a filling defect in its anterior portion (Fig. 9).

A right frontotemporal craniotomy was then done (by J.L.P.) A vertical incision was made and the right lateral ventricle was opened at the anterior horn. A brownish-red glistening mass in the anterior 3rd ventricle protruded through the right interventricular foramen. The mass was a colloid cyst of the 3rd ventricle, which was adherent inferiorly to the choroid plexus.

The capsule was incised and thick gelatinous material was aspirated. The collapsed capsule was then removed.

Course. The patient withstood the operative procedure well and she was discharged on the 12th postoperative day in a markedly improved condition.

When last heard from on Dec. 31, 1951 (6½ years after operation), she reported that she had had no recurrence of headaches and she was working as a private nurse.

Comment. The clinical impression following examination was that the mass was a midline cerebellar tumor, probably hemangioma. This case illustrates the excellent result that can be obtained following removal of a colloid cyst of the 3rd ventricle. Without the aid of ventriculography, proper management of this case would have been difficult.

SYMPTOMATOLOGY

Symptoms of colloid cysts of the 3rd ventricle are produced by an obstruction at the interventricular foramina. Stookey[26] suggested that the severity of the paroxysms might be the result not only of sudden obstruction of the lateral ventricles but also of compression of both veins of Galen, with production of choroidorrhea and acute venostatic edema. The localizing symptoms, when present, are caused either by direct pressure of the cyst on adjacent nerve tissue or, as suggested by Jefferson and Jackson,[13] by hydrocephalic distention of the 3rd ventricle which impairs the blood supply of the cell groups or, less probable, exerts tension on tracts. Larson[15] added that a possible cause of symptoms might be rupture of the cyst into adjacent nerve tissue.

Headache was present as the first symptom in all but 1 case in our series. In Case 2, diminution of libido antedated the onset of headache by 11

months. The duration of symptoms varied from 1 to 11 years. One patient complained of headaches for "many years." The headaches were associated with vomiting in 5 cases. Other associated symptoms were paresthesia (2 cases), "gas in his stomach" (1 case), scintillating scotomata (1 case) and "dizzy spells" (1 case). The headaches, which were intermittent and usually sudden, were referred to any or all areas of the calvarium, from the suboccipital to the frontal regions. In 1 case, the symptoms were characterized as being just as abrupt in recession as in onset. There were no striking instances of any postural nature of the headaches, such as had been described in many reported cases, but this was suggested by some complaints. Sudden rapid movement caused dizziness and blurring of vision in 1 case (Case 4) while in another, pain resulted when the patient turned her head and quick movements caused occasional vertigo with rotation of objects (Case 6).

Dizziness was present in 5 cases and was usually described as occurring in "spells." It was associated with falling in 2 cases. Mental changes were evident before examination in 2 cases; one of these patients (Case 3) had been referred to the clinic after confinement in a mental hospital. Symptoms of blurring of vision, present in 3 cases, were of recent onset when compared with the appearance of the first symptom. One patient had what was described as "generalized tremor like a convulsion and with frothing" after which there was a period of disorientation, amnesia and confusion. Other complaints encountered in single cases were pulsating tinnitus, swishing noise in the ears, bulimia, scintillating scotomata, diarrhea, and epileptiform seizures.

Complaints referable to the lower extremities, and recorded as disturbance of coordination, were present in 3 cases, but there were unusual features in each case. In Case 4, intermittent subjective incoordination of the legs without outward evidence of gait abnormality was a complaint. In Case 3, after episodes of "tremor," the patient was unable to support himself on his feet. In Case 1, unsteadiness in walking was complained of for 6 months but manual dexterity remained normal and at the time of examination ataxia could not be demonstrated. Recurrent episodes of sudden and transient extreme general weakness, and weakness of one or both legs were present in 2 cases (Cases 5 and 7). These transient attacks were striking features in some reported cases. It was noted by Jefferson and Jackson[13] in 2 of their cases, as well as by Hassin[12] and by Shannon.[25]

The impression gained from perusal of our clinical records is that in the early part of the anamnesis, the disturbances were usually intermittent, often with intervals that permitted previous normal activity. The symptom-free intervals tended to shorten progressively just before the patient sought medical aid. The various reviews on the symptomatology have been in general agreement. Headaches influenced by changes in posture, almost uniform suddenness of attacks and intermittency with relatively symptom-free intervals have been the features emphasized.[11,16,29,31] There were no clear-cut instances of postural headache in our series, but in 2 cases there were related

complaints. Intermittency and relatively comfortable periods were present in every instance.

OBJECTIVE FINDINGS

At the time of examination, 2 patients were mentally aberrated to a degree that made tests that required cooperation of the patient unsatisfactory or unreliable. One patient was mildly euphoric. Papilledema was present in 6 cases; in 2 of the 6, it was associated with optic atrophy. The pupils were unequal in 1 case. There was nystagmus in 5 cases; in 1 of these, a slight limitation of upward conjugate movement of the eyes was noted. There were no gross visual field defects; in 1 case in which an upper homonymous partial quadrantic defect with general visual field constriction was found, cooperation was unreliable. A unilateral lower facial weakness was present in 2 cases, unilateral ptosis in 1 case and questionable anosmia in 1. Unequal tendon reflexes with a positive Babinski sign were noted in 1 case, a one-sided equivocal Babinski sign was present in 3 cases and an unsustained bilateral ankle clonus was found in 1 case. Slight hypotonia was present in 2 cases and moderate resistance to passive motion of the lower extremities was found in 1 case. Swaying in the Romberg position was noted in 1 case and falling to the left in 1. In Case 1, in which the clinical diagnosis was a cerebellar lesion and in which a suboccipital exploration was undertaken with normal findings, tremors and dysmetria were observed in the finger-to-nose and heel-to-shin tests. These were more pronounced on the right side; adiadochokinesia was also present on the same side. The fine, rapid tremor of the fingers observed in Case 5 was not interpreted as ataxia, but rather as suggestive of chronic alcoholism.

Other positive features in single cases were endocrine changes characteristic of hypopituitarism, unilateral asynkinesia, cranial bruit, mild exophthalmos and slight unilateral hypesthesia over the extremities.

In all our cases there were definite evidences of chronic intracranial hypertension by the time the patients were admitted to the clinic. As McLean[17] pointed out, many signs referable to nerve structures around the 3rd ventricle probably can be found on examination of "negative" histories, but they are of definite localizing value only when they occur before the onset of pressure symptoms. When present, nevertheless, they should serve to attract attention to this region.

In 5 of our cases, the cranial vault showed well marked convolutional atrophy. In 1 instance (Case 4) diastasis of the sutures was present. The pineal shadow was displaced to the left in 1 case (Case 5). The right lateral ventricle in this instance was larger than the left.

Changes in the sella turcica may sometimes be indicative of colloid cysts of the 3rd ventricle. According to Kopylov,[14] the sellar changes in hydrocephalus secondary to obstruction of the aqueduct at the tentorial level differ from those secondary to obstruction in the posterior cranial fossa. In the former, the dorsum sellae and posterior clinoids have a tendency to deviate posteriorly while in the latter, the height of the dorsum undergoes

more changes and the posterior clinoids have a tendency to deviate anteriorly. The mechanism of the production of these findings is discussed by Kopylov.

In 4 of our patients, there was erosion or flattening of the posterior clinoid processes; in 2 of the 4 patients the posterior clinoids tended to deviate posteriorly and in 1, anteriorly. In the other 3 patients the sella was normal. In 1 patient (Case 3), a tentative diagnosis of a colloid cyst was made on the basis of a long history of intracranial pressure in addition to the roentgenologic evidence of eroded and posteriorly deviated posterior clinoid processes. However, it is doubtful that these findings would ever supplant ventriculography as a method of making a positive diagnosis.

The ventriculograms should have been diagnostic in all of our cases, but in Case 1, a suboccipital exploration was done because of misinterpretation of the ventriculogram. In 4 cases, the 3rd ventricle did not fill with air; in one of these, a filling defect between the ventricles was visible in the anteroposterior view. In 2 cases, partial filling of the anterior 3rd ventricle permitted visualization of the tumor shadow. In 1 case, some air was visible in the 3rd ventricle in the postero-anterior view, but was not demonstrated in the other views nor in the sagittal laminagram.

When a filling defect cannot be demonstrated in the presence of dilated lateral ventricles, a thorough pneumographic study of the 3rd ventricle is indicated. Dandy[5] stated that absence of air in the 3rd ventricle is the most common finding with a tumor of the 3rd ventricle, but it cannot be assumed to indicate a tumor in this cavity unless all the ventricular fluid has been replaced by air. Should a doubt remain, even after manipulation of the head, encephalography may be performed as described by Davidoff and Dyke.[7] Films are taken with the patient in the vertical position. If air enters the 3rd ventricle, the fluid level appears interrupted, with a straight or concave margin in the presence of a tumor in the anterior 3rd ventricle. Davidoff and Dyke stated that this is the most conclusive evidence of a tumor within the 3rd ventricle and may be supplemented by evidence of displacement of sulci and abnormalities of the basal cisterns. Twining[28] described an improved method of filling the 3rd ventricle by increasing the tilt of the x-ray table in addition to elevating the shoulder and hyperextending the head; lateral roentgenograms are then taken. With this technic, the ventricles need not be completely emptied. In a recent paper, Epstein and Davidoff[9] recommended the use of laminagraphy as a routine procedure in pneumoencephalography when visualization of midline structures was necessary. They believed that in an appreciable number of patients the visualization was sufficiently improved so that definite diagnostic conclusions could be reached which otherwise might have been doubtful or overlooked. In other cases, the midline structures could be visualized on the laminagrams and not on the encephalograms. Laminagrams were made in 2 of our cases. In Case 5, an outline of the cyst was demonstrated and in Case 6, the 3rd ventricle showed no filling.

In the review of the air studies in this series, the broad foramina of Monro were constant. The disparity of size between lateral ventricles and the 3rd ventricle is also important. If air is visible in the posterior 3rd ventricle and the ventricle is of normal size and the anterior third is not visualized or poorly visualized in the presence of a broad foramen of Monro, a colloid cyst should be suspected.

Thus, there are several methods for pneumographic investigation of the 3rd ventricle, any one or more of which may have to be used in resolving the pneumographic diagnosis. This is particularly important inasmuch as, in the condition under discussion, all that can usually be said is that the patient has increased intracranial pressure without localizing signs.

OPERATIVE PROCEDURE AND RESULTS

Previous experience in the operative removal of colloid cysts of the 3rd ventricle has been thoroughly reviewed by Weinberger and Boshes[29] in 1943. They collected 17 cases of surgical recoveries up to that date; their series did not include several reported cases.[2,3,13] Dandy, who had tried three different surgical routes in his early cases reserved a transcallosal approach for tumors that extended posteriorly in the 3rd ventricle, and stated that the frequency with which postoperative epilepsy occurred when the transcortical route was employed was a drawback not encountered when the former method was used.[21] However, the latter route has been used exclusively in all the cases reported since the publication of Dandy's[6] monograph in 1933.

In our series, a vertical transcortical incision into the right lateral ventricle was sufficient for exposure in 6 cases (Fig. 2); a small cone of cortex was removed in 1 case. The horizontal incision, preferred by others, had been designed to avoid the contusing effects of direct retraction of the motor area. This objection was largely overcome in Case 6. One of us (J.L.P.) utilized the simple expedient of positioning the patient as for a pituitary operation which permitted the posterior margin of the cortical incision to fall away without direct retraction. With an assistant retracting the anterior margin forward, the operator was left with both hands free.

Partial evacuation of the cyst was necessary in 6 cases before it was delivered from its bed; in Case 6, the cyst was removed intact. As in one of our cases, the cyst occasionally may be irregular, and attempts to remove it intact should be reserved for instances in which, after adequate visualization of the cyst in its entirely, it is believed that removal can be effected without unnecessary trauma. To acquire enough room for dissection, the interventricular foramen was enlarged anteriorly, medially or posteriorly towards the septum pellucidum in all our cases when a perforation was present. In 1 case, both fornices were cut transversely for adequate exposure. In evacuating the cyst, a twofold purpose is accomplished: additional space is obtained and the cyst wall can be grasped with a pair of forceps to aid in the dissection.

In 2 subsequent patients, not included in this report, it has been of

value to incise the cyst and to remove all the inspissated colloid material as an initial step. The stalk of the cyst may then be coagulated with the two-point coagulation and the cyst wall excised without disturbance to the hypothalamic region and with a minimum of retraction.

In all our cases, the cyst was removed in one stage, the craniotomy usually being done immediately following the ventriculographic study. In Case 6 the craniotomy was undertaken 2 days after the ventriculographic diagnosis was made. During the 2-day interval, continuous ventricular drainage was instituted according to the technic described by Poppen.[22] Previous reports[29] have shown that fatalities are particularly prone to occur when the cyst is not removed promptly after an air study.

At operation, one of the technical difficulties encountered was the tendency of the cortex to collapse and fall away from the dura mater when the hydrocephalic ventricle was opened. Retraction, designed to prevent this difficulty, caused subependymal bleeding in Case 4. Before replacing the bone flap, the ventricles were filled with normal saline solution in each case to prevent the cortical collapse. As an added procedure in Case 7, saline solution was injected into the ventricle through a catheter inserted during ventriculography while air was allowed to escape through another catheter, placed for this purpose in the subdural space at the site of the cortical incision.

In Cases 1 and 3 the bone flaps were re-elevated 33 and 15 days, respectively, after removal of the cyst. Both re-elevations were done for presumed clot. In Case 1, the 3rd ventricle was found to be obstructed by old blood and adhesions of the septum pellucidum. In Case 3, considerable edema of the tumor bed was the only finding.

The two conditions are difficult to differentiate unless frank hemorrhage is revealed in the ventricular fluid. McLean suggested that the patient lie face downward for several periods daily with the hope that a small movable clot would be dislodged intermittently until absorption could take place. In the early postoperative period, continuous ventricular drainage may be utilized to prevent the effects of ventricular obstruction.

There were 2 operative deaths in this series. In Case 1, the patient survived 34 days after removal of the cyst and died during hyperthermia, which developed within 24 hours after re-exploration for a 3rd ventricle clot. In Case 6, a fairly satisfactory postoperative course was suddenly terminated 9 days after operation, death occurring within minutes after the onset of unexplained cyanosis. In the immediate postoperative period, transient hemiplegia or hemiparesis was noted in 3 cases. In 6 cases, varying degrees of transient impairment of consciousness ranging from disorientation to deep coma were noted; however, progressive improvement was the rule. In the 1 case in which there were periods of coma, a clot in the 3rd ventricle was found at reoperation. In Case 3, convulsions occurred on the 6th postoperative day.

Of the 5 operative recoveries, 1 patient died almost $4\frac{1}{2}$ years after opera-

tion; the other patients were known to have been alive from 2½ years to 11½ years after operation. In 3 patients convulsions developed. The seizures were uncontrolled and were probably the cause of death in 1 case (Case 3). This patient had had convulsions before and shortly after operation; in addition, frequent spells of uncontrollable rage and a postictal rise in temperature were observed. Two other instances of late postoperative convulsions were mild and readily controlled; the seizure-free periods were 2½ years in 1 case and 4½ years in the other at the time of the last follow-up.

Headaches were relieved completely in 4 patients; in 1 case, the relief was striking, although occasional headaches were associated with the seizures (Case 3). In the same patient, the bulimia that was noted before operation was absent afterward. A memory difficulty, the usual complaint in the early postoperative period in almost all the cases, improved progressively, but appeared to persist to a slight degree for even as long as 8½ years after operation (Case 4). Objectively, no evidences of any significant neurologic defects could be found in 3 patients who were examined 2 weeks to 10 months after operation.

SUMMARY

The detailed clinical histories of 7 cases of colloid cyst of the 3rd ventricle are recorded. All cases were verified by surgical procedures. There were 2 operative deaths. Various aspects in the diagnosis, management and outcome of these cases are described as they were encountered. The mortality in this series is too high for favorable benign lesions. Correct interpretation of the ventriculograms and minimal surgical trauma should make removal possible with slight risk. With the correct diagnosis the cyst can readily be removed through a well placed trephine opening if the cyst contents are evacuated and the entering vessels from the choroid plexus coagulated with the two-point coagulation, as was done by one of us (J.L.P.) in a subsequent case. This procedure obviously should be instituted only in the patient in whom the ventriculographic studies demonstrate a small well rounded mass in the foramen of Monro, making the diagnosis certain.

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POST-TRAUMATIC PULSATING EXOPHTHALMOS

CAUSED BY PERFORATION OF AN ERODED ORBITAL ROOF BY A HYDROCEPHALIC BRAIN

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THE relation between certain vascular anomalies and pulsating exophthalmos has been known for many years. The first fundamental observations were made by Travers (1809), Dalrymple (1815), Baron (1836) and Nelaton (1856). However, a long time elapsed before the importance of abnormal cranio-orbital communication as a cause of pulsating exophthalmos was clearly recognized. The publications of Moore,[7] LeWald[5] and Wheeler[11] directed attention to pulsating exophthalmos in individuals with congenital defects of the superior or/and posterior orbital walls. In several of their cases the lesion was associated with diffuse neurofibromatosis.

Abnormal communication between the cranial cavity and the orbit may exist through preformed openings (foramina and fissures) and through congenital or acquired defects in the bony orbital walls. The acquired defects are traumatic or caused by disease. Strandberg[10] divided the latter in order of frequency into: bone erosion caused by aneurysms, malignant tumours, inflammatory processes, angiomas, von Recklinghausen's disease, Paget's disease, and internal hydrocephalus. From the literature it appears that in such cases of pulsating exophthalmos the intracranial pressure may be normal.

Van der Hoeve[4] published a case in which spontaneous pulsating exophthalmos was thought to be the consequence of internal hydrocephalus. According to him the increased intracranial pressure had caused a widening of the optic foramen, followed by a herniation of the brain through this opening.

It is most remarkable that operative removal of large parts of the orbital walls, which is inevitable in transcranial extirpation of intra-orbital tumours, is practically never complicated by pulsating exophthalmos. In his monograph on orbital tumours Dandy[2] stated: "The thought doubtless occurs whether pulsating exophthalmos does not follow removal of the orbital roof. I have looked carefully for such a sequela but have never found it, despite the fact that pulsating exophthalmos is known to occur when the orbital roof is congenitally absent."

Wheeler[11] considered that the nature of the tissues filling the orbital defect determines whether the cerebral pressure and pulsations will be transmitted to the orbital content or not. After operative removal of the orbital

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roof newly formed tissue replaces the defect and in the course of time this layer becomes firm connective tissue, acting as an effective barrier against intracranial pressure and pulsations.

In the writer's experience, operative removal of the orbital roof was followed by pulsating exophthalmos in one instance. In this case the intracranial operative removal of the orbital roof was part of the treatment of a cranio-orbital injury. The postoperative course was complicated by meningitis and postmeningitic hydrocephalus. During the period of increased intracranial pressure pulsating exophthalmos developed, but it subsided completely after the successful relief of the hydrocephalus.

It therefore seems reasonable to distinguish the mechanisms of pulsating exophthalmos in cases of abnormal cranio-orbital communications in two categories: (1) A lowered mechanical resistance of the cranio-orbital barrier, which replaces the defect, either as a result of a developmental disturbance or local disease. It does not seem to be of fundamental importance that the intracranial pressure should be increased at the time of the examination. (2) The increased intracranial pressure and pulsations are the main factors against which the reparative processes replacing the orbital defect are only partly effective. There is a direct relation between the pulsating exophthalmos and increased intracranial pressure, viz. lowering of the intracranial pressure makes the exophthalmos disappear.

It is the second group that has aroused our special interest. In 2 of our cases the pulsating exophthalmos that occurred after cranial injuries resulted from herniation of a hydrocephalic brain through a perforation in the orbital roof.

In chronic internal hydrocephalus the thinning of the orbital roof from the pressure of the brain may be considerable. Fig. 79 in Dorothy Russell's[9] report on hydrocephalus shows marked erosions at the base of the skull in such a case; the bone of the orbital roof was so thin that a hole was accidentally made with the point of the forceps whilst stripping the dura, exposing underlying adipose tissue. If, for instance, such a person falls and strikes his head against something, the brain is consequently set into motion. If the movement of the skull is stopped, the brain continues its movement until this is resisted by certain parts of the skull. If the circumstances of the accident determine a rotatory movement of the brain towards the roof of the orbit, the latter yields to the impact, which means perforation of the roof and herniation of the brain into the orbit. It seems also possible that the fracture of the orbital roof is caused by vibrations, set up by the injurious forces. Sooner or later the fractured part of the orbital roof gives way to the high pressure of the intracranial content and intra-orbital herniation of the brain follows.

CASE REPORTS

Case 1. #N. 2080. Y.M., a female 24 years old, was admitted on July 22, 1946. In December 1943 she had been hit by a motorcar and was admitted to an Indonesian hospital with a basal fracture of the skull. She remained unconscious for 4

days. The right eye was displaced downwards and during subsequent years a progressive pulsating exophthalmos developed. Other disturbances such as amenorrhoea, bradypsychia and a certain stiffness of gait also remained.

Examination. There was bilateral papilloedema. The right eye protruded and was displaced downwards. Upward movement of this eye was limited. The cornea and sclera appeared normal; there was no chemosis. On palpation a soft mass above the eye was felt through the upper eyelid. No vascular murmur could be detected. There was dorsi-extension of the great toe on both sides (Babinski). Sugar tolerance test showed a normal fasting level, but the peak of the curve was 204 mg. per cent; a normal value, however, was restored within 2 hours. There was no abnormal reaction of the blood sugar to adrenalin and insulin. B.M.R. was −23 per cent. Water balance was normal.

FIG. 1. *Case 1.* Depressed fracture with perforation of the right orbital roof.

Roentgenograms of the skull showed increased convolutional impressions. The cranial vault was very thin. The sella was enlarged with widening of the entrance. There was a depressed fracture of the right orbital roof (Fig. 1).

Ventriculography on Aug. 16, 1946, revealed a pronounced internal hydrocephalus and atresia of the aqueduct. The right frontal horn extended downwards into the right orbit (Fig. 2).

Operation. A right frontal craniotomy was performed. The dura mater was incised after ventricular tap. On elevating the tip of the frontal lobe, the prolapse into the orbital roof became visible. As this was irreducible, the neck was cut, whereby the ventricle was inevitably opened. The frontal lobe was then elevated, and using the procedure of Stookey and Scarff the lamina terminalis and the floor of

the 3rd ventricle were perforated to relieve the obstructive hydrocephalus. The orbital roof was exposed extradurally, the hernial formation was excised and the depressed bone was removed. The orbital roof was covered with an osteoperiosteal bone graft obtained from the tibia. The dural defect was closed with a fascia lata graft and the wound was closed.

Course. Postoperatively the eye did not protrude and there were no pulsations, although there was still some downward displacement of the eye. Convalescence was slow because of the patient's attitude of indifference and inactivity, which had been apparent before operation.

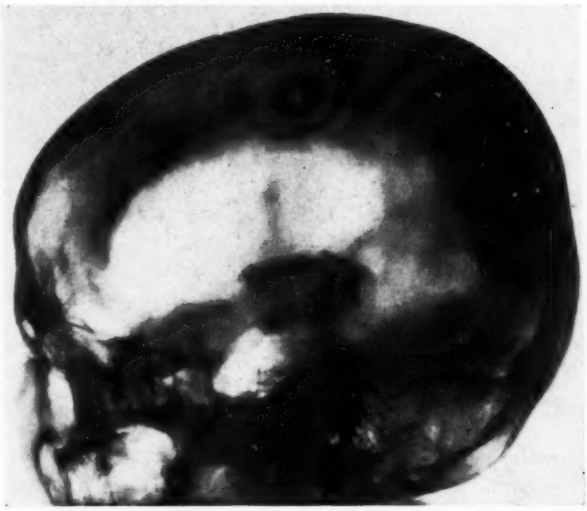

FIG. 2. *Case 1.* The frontal horn of the dilated right lateral ventricle shows an outpouching through the right orbital roof. The occluded aqueduct tapers to a point.

Case 2. #N. 8760. J.C. de K., a male 25 years old, had fallen from his bicycle on Aug. 31, 1951 and knocked his head against the tramway-rails. He visited his doctor and then went back to work. A few hours later, however, he began to feel ill and was admitted to hospital, where a diagnosis of basal fracture of the skull and a pulsating exophthalmos of the right eye was made. On Sept. 2, 1951, he was transferred to the neurosurgical ward as it was suspected that an arteriovenous fistula was the cause of the pulsating exophthalmos.

Examination. The patient complained of attacks of headache and vomiting. There was an extreme pulsating exophthalmos of the right eye, with chemosis and keratitis. The eye was deviated downward. All eye movements were limited, especially elevation. A soft mass was found protruding behind the upper eyelid. This mass could be reduced by finger pressure and it did not seem to be adherent to the eyebulb.

Roentgenograms of the skull revealed enlargement and erosion of the sella. The occipitomental view showed a fracture with perforation of the anterior part of

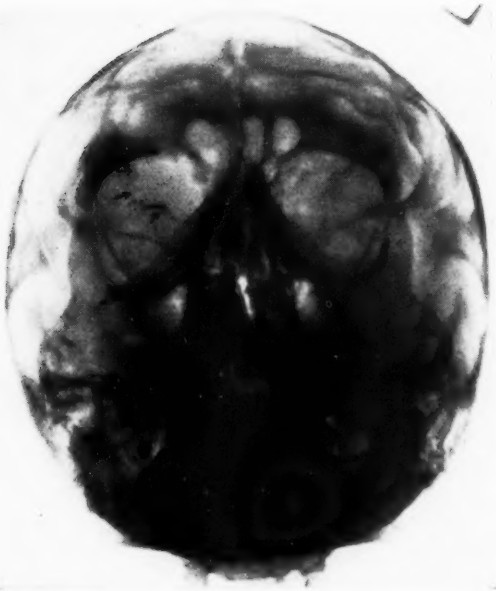

FIG. 3. *Case 2.* The occipitomental view shows a fracture with perforation of the right orbital roof (arrows).

the right orbital roof (Fig. 3); the occipitofrontal view did not show these orbital changes.

Ventriculography was done with a small quantity of air according to the method of Ziedses des Plantes.[12] Both lateral ventricles were dilated; after the "saltus" the air collected in a widened posterior part of the 3rd ventricle; the anterior part of the aqueduct was filled and it was not funnel-shaped. The exophthalmos could be reduced by lowering the ventricular pressure.

1st Operation, Sept. 3, 1951. Our first attempt was to relieve the hydrocephalus. As there was some doubt about the nature of the obstruction the approach was made by a suboccipital craniotomy. The arch of the atlas was removed in order to free the incarcerated tonsils of the cerebellum. The cerebellum appeared completely normal and the 4th ventricle was intact. A No. 10 catheter was left in the aqueduct for some days with the other end of it guided through the muscles and skin, and connected with a receptacle. The wound was closed.

Course. After the operation there was only a slight degree of exophthalmos with faint pulsations.

On Sept. 6, 1951 there was no outflow from the catheter. The intracranial pressure was high (35 cm. H_2O) and there was pronounced exophthalmos. The catheter was withdrawn and the ventricle was tapped. The patient became soporose. Arterial hypertension, bronchial hypersecretion and profuse sweating developed.

2nd Operation. The aqueduct and the 4th ventricle were found to be blocked by blood clots, which were removed. Lavage of the ventricles was performed whereupon the CSF circulation seemed to be re-established.

Course. The ventricular pressure remained normal, and the exophthalmos completely disappeared. However, the keratitis, present on admission, had resulted in panophthalmia, necessitating evisceration of the bulbus on Sept. 14, 1951. Thereafter convalescence was uneventful.

3rd Operation. Two months later, on Nov. 12. 1951 right frontal craniectomy was done. The cerebral prolapse into the orbit appeared to be irreducible. The prolapse was divided at the neck and the intra-orbital part was excised. The orbital roof was covered by an osseous graft from the ilium and the dural defect was closed by a fascia lata graft.

Course. The patient left hospital 2 weeks later with a normal intracranial pressure. No pulsations could be felt through the orbit.

DISCUSSION

Although nowadays it is generally known that pulsating exophthalmos may be produced by different lesions, many clinicians are still inclined to attribute all cases of post-traumatic pulsating exophthalmos to a carotid-cavernous aneurysm (Rowbotham;[8] Mock[6]). In discussing the differential diagnosis, Mock stated: "A history of trauma will nearly always be obtainable for the diagnosis of a carotid-cavernous arteriovenous aneurysm." Our 2 cases show that traumatic pulsating exophthalmos may be caused by another condition, namely perforation of a previously eroded and thin orbital roof by the impact of a hydrocephalic brain. These 2 cases represent 28 per cent of the total number of cases of post-traumatic pulsating exophthalmos observed in our hospital during the last 10 years. In our other 5 cases the cause was an arteriovenous aneurysm.

DIAGNOSIS

In order to avoid a diagnostic error in cases of post-traumatic pulsating exophthalmos, it is very important to make an X-ray examination of the skull, especially of the orbital roof. Sometimes the defect may not be visible in the occipitofrontal view of the skull, whereas it is usually evident in the occipitomental view (see Case 2). Post-traumatic pulsating exophthalmos caused by hydrocephalus and perforation of the orbital roof may have the following features in common with the exophthalmos associated with arteriovenous aneurysm:

1. The pulsations of the eye disappear or diminish on compression of the carotid artery.
2. Chemosis of the affected eye (Case 2) may be present.
3. Papilloedema. It should be noted, however, that in Case 1 papilloedema was present not only on the affected side, but was bilateral.

In post-traumatic pulsating exophthalmos caused by herniation of hydrocephalic brain there are no vascular thrills or murmurs. The protruding brain may be visible or palpated behind the upper eyelid. The exophthalmos diminishes after a tap of ventricular fluid. The skull may show typical signs of chronic hydrocephalus. Ventriculography serves as a most valuable aid in the diagnosis.

It should also be pointed out that the patient may never have noticed a pre-existing pulsating exophthalmos associated with a congenital orbital defect and that his attention was drawn to it only after the trauma. This may give a wrong impression to the doctor who examines the patient for the first time after the trauma.

Treatment. The first objective should be to relieve the internal hydrocephalus, either by removal of the cause of obstruction or by palliative procedures, depending upon the circumstances. Once the intracranial pressure has been reduced, the treatment of the cerebral hernia with plastic repair of the orbital defect will not be found difficult.

Failure to recognize this condition may lead to erroneous interventions, such as carotid ligation. Gardner[3] reported a case of post-traumatic unilateral exophthalmos caused by outpouching of a hydrocephalic brain through a fractured orbital roof. The patient suffered from a cerebellar tumour. A frontal craniotomy was performed, as a diagnosis of meningioma of the sphenoid ridge with invasion of the orbit had been made. The operation revealed the true nature of the lesion, and further examination led to the surgical treatment of a large cerebellar cystic haemangioblastoma, which had caused the obstructive hydrocephalus.

Corbella[1] described a 26-year-old patient with post-traumatic exophthalmos. Enucleation of the eye was followed by an enormous intra-orbital protrusion, which became visible in the region of the upper eyelid. This protrusion consisted of herniated frontal lobe containing a diverticulum of the ventricle. Roentgenograms of the skull showed convolutional markings and separation of the sutures. Ventriculography demonstrated an internal hydrocephalus. An effort to reduce the cerebral herniation by a transfrontal approach failed and the patient died 2 days later. Corbella thought that the traumatic brain lesion was the cause of the hydrocephalus. However, it seems more probable that the changes in the skull evident in the X-rays were not the result of hydrocephalus of 3 months' duration, as reported by the author, but were caused by chronic pre-existing hydrocephalus. From this point of view Corbella's case might be considered in the category of patients with chronic hydrocephalus, in whom a cranial injury results in traumatic rupture of the orbital roof with outpouching of the brain into the orbit.

SUMMARY

Although abnormal cranio-orbital communications are well-known causes of pulsating exophthalmos, there is still a general tendency to attribute all cases of post-traumatic pulsating exophthalmos to vascular lesions. The author, however, points to a special type of post-traumatic pulsating exophthalmos that occurs in patients in whom the orbital roof has become thin and eroded as a result of chronic hydrocephalus. The secondary movements of the brain and of the ventricular fluid, set up by the injury, may force the brain through the weakened orbital roof, or the vibrations, set up by the injurious forces, may result in a fracture of the orbital roof, present-

ing a locus minoris resistentiae to the expansive forces of the hydrocephalic brain. If the patient had had no complaint indicative of chronic hydrocephalus prior to the injury, the danger of a diagnostic error is imminent. Every patient suffering from post-traumatic pulsating exophthalmos should be subjected to a thorough X-ray examination of the orbital roof in different views. Other valuable diagnostic aids are the roentgenological signs of chronic hydrocephalus (ventriculography included) and the relation of ventricular pressure to the degree of exophthalmos.

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CONTROLLED HYPOTENSION*

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HYPOTENSION has long been considered synonymous with shock, and hence its occurrence during surgical procedures is looked upon with fear or apprehension. However, blood pressure readings below normal need not be associated with shock. In recent years arterial hypotension has been utilized in attempts to reduce the loss of blood during operations. By inducing hypotension, it has been possible to perform certain operations more easily, more quickly, and with less risk, and to undertake others that would be impossible without this technique.

If it is true that a fall of blood pressure on one hand means shock, and on the other can be induced intentionally with little danger to the patient, it seems that there must be a fundamental difference between these two types of hypotension.

It will therefore be necessary to consider now some points concerning the factors that maintain blood pressure and to discuss the mechanisms that come into play when hypotension supervenes either because of shock or in consequence of deliberate action on the part of the physician.

PHYSIOLOGY OF BLOOD PRESSURE

Arterial blood pressure is maintained through a number of factors, the chief ones of which are: (1) cardiac output; (2) peripheral resistance; (3) amount of blood in the arteries; (4) blood viscosity; (5) elasticity of the arterial walls. Of these the first four factors contribute in regulating systolic blood pressure, while the last factor is chiefly instrumental in the maintenance of diastolic pressure. Any change in any one of these factors, sufficiently great or sustained to overcome the compensatory powers of the organism, will effect a rise or fall of blood pressure.

In circumstances that produce shock, such as for instance hemorrhage, the immediate reaction is a constriction of the arterioles, or more precisely of the metarteriolar sphincter mechanism. This represents an attempt at compensation which tends to maintain the blood pressure at its previous level. Trying to maintain the circulating volume at an adequate level, fluid

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is next shifted from the interstitial spaces into the blood stream, thus increasing the total plasma volume. When this takes place, one talks of "compensated" or better "incipient" shock. However, if hemorrhage continues without adequate replacement or if anoxic or stagnant hypoxia is superimposed, then arteriolar and capillary dilatation and stasis will develop. The consequence of this is a reduction of venous return and a fall of cardiac output with subsequent failure of compensation, hypotension, and the development of "frank" shock. If shock is not promptly corrected, the hypoxia increases until finally the integrity of the capillary wall is lost with such irreparable hypoxic damage as to make the shock "irreversible." The phenomenon of capillary stasis is demonstrated clinically by the increase of capillary refill time. The mechanism of hypotension as described pertains to cases of so-called "oligemic shock."

Another mechanism by which hypotension may be produced is one in which primary dilatation of the arteriolar bed occurs. Thus, by augmenting grossly the vascular bed and by decreasing peripheral resistance, a fall in arterial blood pressure ensues. In this kind of hypotension, capillary circulation should remain adequate, as can be demonstrated by very prompt refill time. Hence, venous return remains satisfactory and cardiac output is only moderately reduced. This is the mechanism acting in the rather badly termed "neurogenic shock," and is encountered clinically, for example, during spinal anesthesia.

It is, therefore, the adequacy or inadequacy of capillary circulation that determines whether or not the blood pressure fall is or is not a true shock state.

From the foregoing it is seen that hypotension of the first type associated with tissue hypoxia and leading to tissue changes cannot be tolerated for very long, and must be corrected by all the means at our disposal. Hypotension of the second type, on the other hand, has few deleterious effects, provided capillary circulation remains adequate and sufficient oxygenation of the blood is maintained. If these two prerequisites are followed, no damage will result to such vital organs as the brain, heart, kidneys and adrenals. This has been demonstrated both clinically and experimentally. Admittedly, filtration of urine probably ceases if the systolic pressure in the renal arteries falls below 75 mm. Hg (although this may be too high a value according to the latest investigative work). None the less, the nephron remains undamaged with pressures probably as low as 45–50 mm. Hg and may be even less, so that formation of urine is resumed when the filtration pressure is again reached. Let it be emphasized that this applies only with good capillary circulation and adequate oxygenation. With regard to the coronary arteries, even in individuals with coronary insufficiency, attacks of angina pectoris do not occur if arterial hypotension is induced by arteriolar dilatation, and if arteriosclerotic changes have not reduced the elasticity of the coronary vessels. If we consider the brain, we are more uncertain, as no one knows the minimum pressure necessary to maintain adequate nourishment of this sensi-

tive and highly specialized organ of high metabolic requirements. The problem of adequate cerebral circulation and oxygenation will be discussed in more detail later.

METHODS OF CONTROLLED HYPOTENSION

Hypotension may be induced by a variety of means but some, such as large doses of barbiturates and deep general anesthesia, are obviously undesirable. Three principal methods have evolved in recent years and these will be considered in detail. They are:

1. Controlled arteriotomy and arterial re-transfusion.
2. Preganglionic block by spinal anesthesia.
3. Ganglionic and postganglionic block by means of pharmacological blocking agents.

1. *Controlled Hypotension by Arteriotomy and Re-Transfusion.* This procedure consists essentially of withdrawing blood from an artery until a suitable degree of hypotension is reached, and maintaining this hypotensive level by judicious re-infusion and re-bleeding from time to time.[3,10,16,19] The blood withdrawn is prevented from clotting by the usual methods and is returned to the patient at the conclusion of the operation. Additional blood may then also be given if needed. The blood pressure is maintained at a level insuring a relatively bloodless field, but is never allowed to fall below 80 mm. Hg in a normotensive individual. Correspondingly higher levels must be maintained in hypertensive and arteriosclerotic individuals. There is often a slight rise in blood pressure following withdrawal of the first small amount of blood, no doubt caused by a compensatory arteriolar constriction and increase in plasma volume. If bleeding is still troublesome at the operative site, further blood will have to be withdrawn until the desired level of hypotension is reached or good control of bleeding achieved. Should the blood pressure fall below 80 mm. Hg, re-transfusion of small amounts of blood is started immediately until the pressure again reaches 80 mm. Hg. The average duration of hypotension possible with this method is reported to be 3 hours with a maximum of 6 hours. The amount of blood necessarily withdrawn may be as much as 3000 cc. If the pressure is restored to normal at the conclusion of the operation and not all the blood re-transfused, the remainder is administered by slow intravenous drip, if necessary. The pulse must remain slow at all times. Otherwise re-transfusion must be undertaken immediately.

This method of selective hypotension has been used mainly in the past for cranial surgery, and the advantage claimed for it is better visualization of the operative field as it is not obscured by blood, and visualization is further aided by the reduction of the volume of the brain itself. Various apparatuses have been constructed for the withdrawal and re-transfusion of blood, and are described in the literature.[3,10,16,22] A number of fatalities have occurred with the technique and are understandable, as the mechanism of producing this type of hypotension is by arteriolar constriction and carries

with it all the potential inherent dangers of shock. Frank shock is avoided by maintaining the blood pressure at about 80 mm. Hg, but the margin of safety is relatively small. Another danger is possible damage to the artery and even loss of the artery. Failure of the apparatus may occur and the possibility of air embolism is real. It must be remembered that the power of compensation may be extremely poor in dehydrated and hypovolemic patients. High oxygen concentration must of course be maintained throughout the operation and extra blood must be available to maintain the existing state of incipient shock. At the time this technique was developed, nothing better was available. Probably because of its inherent dangers and its dependence upon all the compensatory powers of the body, this method has only a few proponents and is now being superseded by other less hazardous techniques.

2. *Controlled Hypotension by So-Called Total Spinal Anesthesia.* This method was first described by Griffiths and Gillies in 1948.[11,12,15] The hypotension is produced by arteriolar dilatation resulting from a preganglionic sympathetic block which paralyzes the vasoconstrictor innervation. It is based on a number of experimental observations which should be reviewed briefly. First, it has been shown that the severity of the fall in blood pressure depends upon the number of spinal segments blocked, and that the fall is relatively greater the more cephalad the segments have been blocked. Hence for maximum effect a block as high as the first thoracic segment is necessary. Blocks at higher levels are undesirable and cause no further fall of blood pressure as no sympathetic vasomotor fibres arise above T1.[15] It has also been shown that with blocks higher than T1, death may result from respiratory paralysis caused by paralysis of the phrenic nerves. Cardiovascular failure is a secondary phenomenon[25] that can be prevented by adequate oxygenation. With artificial respiration, the blood pressure can be maintained at 40–60 mm. Hg.[8] Since the advent of spinal anesthesia, this has been demonstrated in a number of cases when the anesthesia has risen above the desired level. In relatively early times, operations were performed above the diaphragm with spinal anesthesia without apparent untoward effects.[18,23,21,27,29] In those operations, it was found that the operative wound was dry, and the breathing was of the diaphragmatic type, indicating that no paralysis of the phrenic nerves had occurred. A point these authors stressed was the necessity of the Trendelenburg position to assure adequate blood supply to the brain.[28]

Griffiths and Gillies revived the concept of utilizing high spinal anesthesia to reduce blood loss during sympathectomies. In so doing, they rely on the concept of "differential spinal anesthesia,"[24] which is based on the fact that the least myelinated nerve fibres are the first to be blocked by anesthetic substances.[17] Hence, by using dilute solutions of anesthetic drugs in the subarachnoid space, it is possible to block sympathetic fibres with a minimum of sensory and motor impairment. Greene has applied the term "hypotensive spinal anesthesia" to this technique.[14] General anesthesia must be used

throughout the operation. The patient is kept in the Trendelenburg position, and as long as capillary circulation remains sufficient and tissue oxygenation adequate, the method is safe. Bradycardia, which often occurred in Gillies' cases, was caused by paralysis of the cardio-accelerator fibres in the upper thoracic segments, and the consequent increase of vagal tone. Cyanosis, they state, was almost invariably caused by inadequate oxygenation, but had to be differentiated from circulatory failure. Respiratory arrest requires artificial ventilation. The breathing is usually slow and largely diaphragmatic. If breathing is spasmodic, supplementary or controlled ventilation is indicated. The original technique described by Gillies for sympathectomy and based on that of Vehrs[28] consists of injecting Procaine (150–250 mg. dissolved in 3–4 cc. cerebral spinal fluid) at L_2, turning the patient on his back and then into a steep Trendelenburg position. Before the pressure falls to extreme limits, the tilt of the patient is reduced to approximately 20° (Trendelenburg). Oxygen administration is started before extreme hypotension supervenes. Within 10–20 minutes blood pressure readings cannot be obtained at the brachial artery and the arterial pulse at the wrist is not palpable. With this method, anesthesia is found over the entire body, but motor paralysis extends only to the first thoracic segment. The phrenic nerve and cranial nerves are unaffected. Within 20–30 minutes respiratory and circulatory function return to normal, the radial pulse becomes palpable, and blood pressure is low but measurable.

This technique was amended later so that the blood pressure is no longer allowed to become imperceptible. Yet, the method as originally described showed the relative safety of the technique. There was only one death in the series of 84 operations (44 patients) reported. Gillies also showed that at most only slight interference with kidney function was demonstrated in his series of cases. Oxygen tension and oxygen saturation are unchanged; only slight alterations occur in pyruvate and lactate levels, indicating that cell oxidation is not affected during spinal hypotension[13] provided adequate pulmonary oxygenation is assured and that the blood volume remains constant.

A more recent technique utilizes either a single injection of Procaine or continuous spinal anesthesia with 0.5 per cent Procaine affecting predominantly the sympathetic component. Any of the other spinal anesthetic agents may be used, but hypobaric solutions are better avoided as their spread cephalad requires elevation of the head, which is undesirable when hypotensive techniques are employed. Anesthesia is maintained by endotracheal nitrous oxide or cyclopropane with ether. Controlled or assisted respiration is used. The patient is in slight Trendelenburg position and blood pressure is controlled in part by elevation or lowering of the legs. The legs thus may be likened to a reservoir of blood available for autotransfusion. A systolic pressure of approximately 50–60 mm. Hg is the aim. Adequate intravenous fluids and medications are given. It remains the anesthetist's choice to use the spinal anesthetic as both hypotensive agent and analgesic agent without other anesthetic supplement (other than oxygen), to use supplementary ni-

trous oxide or sodium pentothal sleep, or to use dilute Procaine for the sympathetic block and general anesthesia for motor and sensory block and maintenance of unconsciousness. The latter is the method of choice, particularly in operations above the diaphragm.

While this method has proven satisfactory and apparently safe in the hands of Gillies and other workers, it too has not achieved widespread acceptance because of its complexity and because it combines the inherent dangers of a spinal anesthetic and those of general anesthesia.

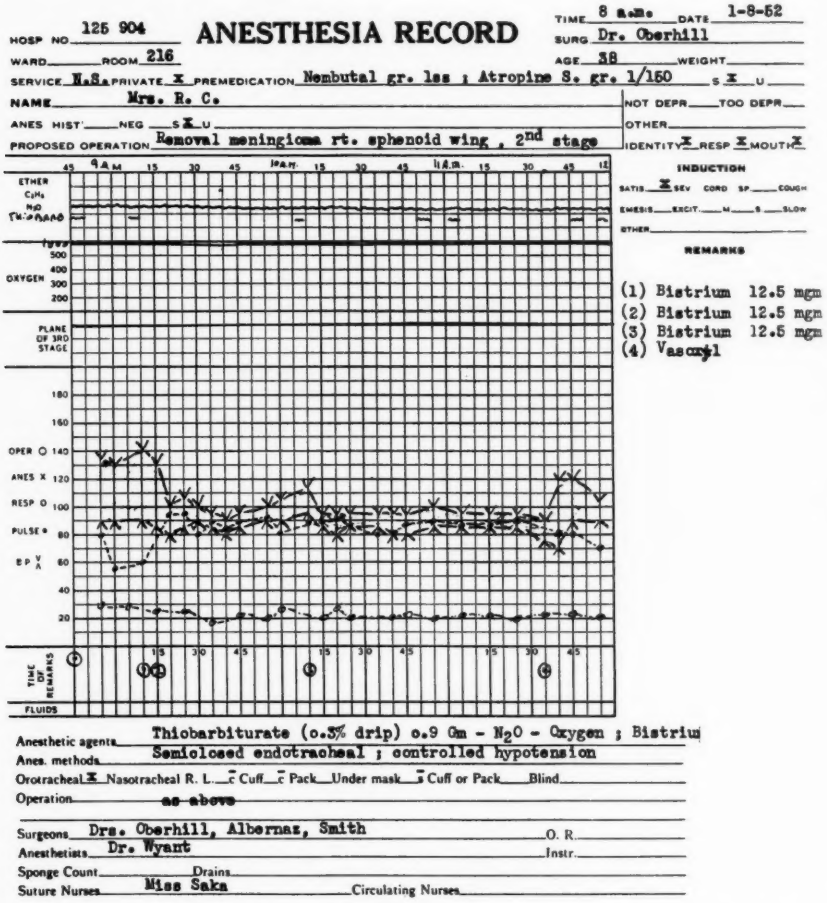
3. *Controlled Hypotension by Means of Blocking Agents.* In search of an antidote for the muscle relaxant Syncurine® (C10, decamethonium), pentamethonium was studied extensively, and it was for a time considered the antidote for C10. It was seen that its administration was accompanied by a marked fall in blood pressure of occasional serious proportions. This hypotension was found to be caused by its blocking of autonomic ganglia.[1,2,23] These observations in due course led to clinical use of the methonium compounds for the treatment of hypertension. In 1950 Enderby[6] suggested the substance may have value in producing controlled hypotension and thus serve as a more practical substitute for Gillies' method of total spinal anesthesia. In his original paper Enderby pointed out that the methonium drugs act by blocking autonomic ganglia, producing an increase in blood flow by arteriolar dilatation and thus a subsequent fall of arterial pressure.

Again, we must consider some of the basic work. Paton and Zaimis[23] established experimentally the absence of changes in liver and kidneys in animals that received large doses of methonium compounds. Shackleton[26] in 1951 reported that a systolic pressure below 60 mm. Hg maintained for $3\frac{1}{2}$ hours caused no alteration in the function of vital organs. It has been established that renal blood flow remains constant if blood pressure is lowered deliberately by means of ganglionic blocking agents during anesthesia.[4]

Enderby's technique[6,7] to obtain a relatively bloodless surgical field is divided into two distinct manoeuvres. The first consists of lowering the blood pressure to 55–65 mm. Hg which he considers the optimum, the other of elevating the site of operation, a manoeuvre to effect what he called "postural ischemia." Davison[5] has pointed out that reduction of blood pressure per se, if caused by diminished peripheral resistance, presumably does not reduce bleeding but rather the hydrostatic effect of posture is the factor of great importance. He states that with a systolic blood pressure of 200 mm. Hg, a foot down tilt of 15° will alter the normal ratio of blood flow to the head, which is $\frac{1}{3}$, to a ratio of 7/24, an alteration of no appreciable significance. But with a pressure of only 30 mm. Hg, the change will be from the normal of $\frac{1}{3}$ to a striking 1/7 ratio. A point may thus be reached, and we don't know precisely what it will be for the particular patient, where elevation of the head, to reduce bleeding during a craniotomy, will cause such interference with cerebral circulation that permanent damage results. This is a most important consideration as the advocates of controlled hypotension by methonium compounds claimed that the postural effect of the technique

is an important part in affording a dry field. Postural ischemia may be employed with safety in operations upon the extremities, abdomen, and chest. Conversely in cranial operations, the technique is by no means free of danger. In fact, there are cases recorded in which decerebration, blindness, or other sequelae followed operations upon the head when postural ischemia was employed in obtaining a bloodless field.

Fortunately, in cranial surgery, an absolutely dry field is not generally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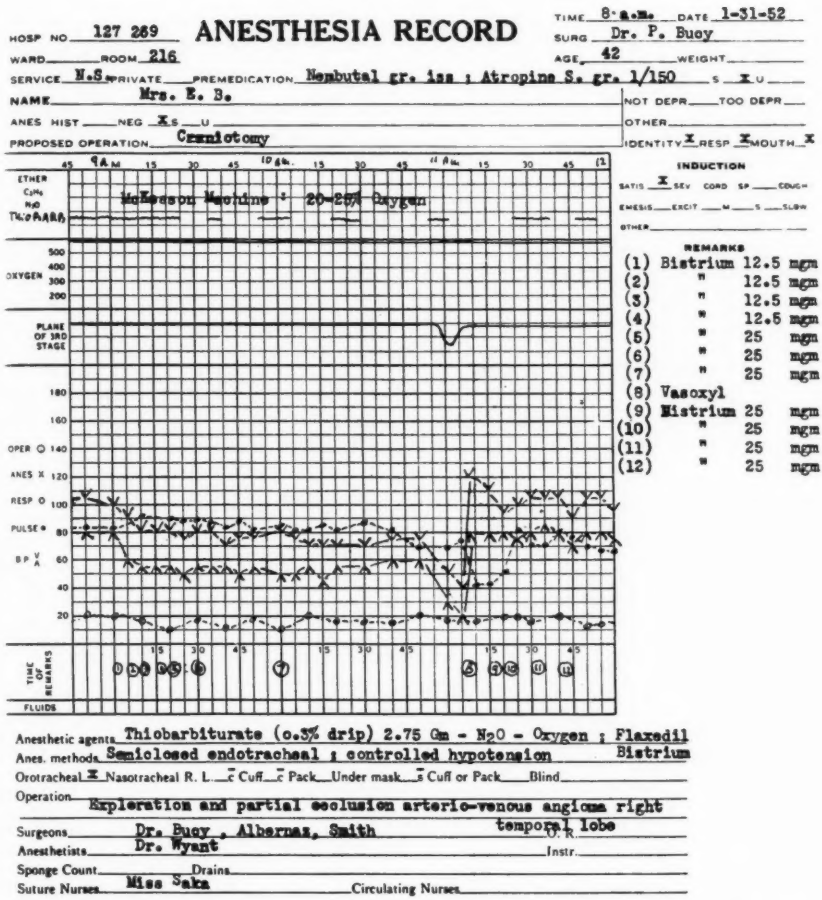
SERVICE N.S. PRIVATE X PREMEDICATION Nembutal gr. 1ss ; Atropine S. gr. 1/150
NAME Mrs. R. C.
ANES HIST NEG S X U
PROPOSED OPERATION Removal meningioma rt. sphenoid wing , 2nd stage
TIME 8 a.m. DATE 1-8-52
SURG Dr. Oberhill
AGE 38 WEIGHT
NOT DEPR TOO DEPR
OTHER
IDENTITY X RESP X MOUTH X
INDUCTION
SATIS X SEV CORD SP COUGH
EMESIS EXCIT M S SLOW
OTHER
REMARKS
(1) Bistrium 12.5 mgm
(2) Bistrium 12.5 mgm
(3) Bistrium 12.5 mgm
(4) Vasoxyl
ETHER
OXYGEN
PLANE OF 3RD STAGE
OPER O
ANES X
RESP O
PULSE •
BP
TIME OF REMARKS
FLUIDS
Anesthetic agents Thiobarbiturate (o.3% drip) o.9 Gm - N2O - Oxygen ; Bistrium
Anes. methods Semiclosed endotracheal ; controlled hypotension
Orotracheal X Nasotracheal R. L. Cuff Pack Under mask Cuff or Pack Blind
Operation as above
Surgeons Drs. Oberhill, Albernaz, Smith O. R.
Anesthetists Dr. Wyant Instr.
Sponge Count Drains
Suture Nurses Miss Saka Circulating Nurses

FIG. 1. 38-year-old woman. Removal of meningioma from right sphenoid wing. Previous attempt at removal had to be abandoned because of excessive bleeding. Tumor was removed with comparative ease and speed under hypotensive anesthesia.

Note that systolic pressure was never below 90 mm. Hg, but narrowing of pulse pressure was marked. Pulse rate remained at around 90. Only small and infrequent doses of Bistrium were needed. Effect was promptly reversed by Vasoxyl.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(6) " 25 mgm
(7) " 25 mgm
(8) Vasoxyl
(9) Bistrium 25 mgm
(10) " 25 mgm
(11) " 25 mgm
(12) " 25 mgm
Anesthetic agents Thiobarbiturate (0.3% drip) 2.75 Gm - N2O - Oxygen ; Flaxedil
Anes. methods Semiclosed endotracheal ; controlled hypotension Bistrium
Orotracheal X Nasotracheal R. L. Cuff Pack Under mask Cuff or Pack Blind
Operation Exploration and partial occlusion arterio-venous angioma right temporal lobe
Surgeons Dr. Buoy , Albernaz, Smith O.R.
Anesthetists Dr. Wyant Instr.
Sponge Count Drains
Suture Nurses Miss Saka Circulating Nurses

FIG. 2. 42-year-old woman. Exploration and partial occlusion of arteriovenous angioma of right temporal lobe.

Note initial slight drop of pressure after first dose of Bistrium, not adequate for surgical needs. After 125 mg. there was some narrowing of pulse pressure with better operating conditions. One hour after last dose sudden marked hypotension occured, but no tachycardia or other signs of shock. Marked reaction to Vasoxyl, then stabilization of blood pressure. No effect from further 100 mg. Bistrium.

required by the surgeon, but only a sufficient reduction of bleeding to facilitate control of bleeding points. In those cases we do not attempt to produce a thoroughly dry field. Clinically, this reduction of bleeding is achieved by producing a moderate fall of systolic pressure without producing a corresponding fall of the diastolic pressure. The result is a diminished pulse pressure. Under these circumstances, capillary oozing is decreased markedly, but larger vessels continue to bleed. These can, however, be controlled with

ease. Furthermore, aneurysms and other vascular lesions show less tendency to rupture and bleed. The technique has not only proved safe, but highly satisfactory to the operating surgeon. Less parenteral fluids are needed and the patient does extremely well postoperatively.

A big disadvantage of the methonium compounds is the degree of uncertainty of response of the patients to these drugs. While prompt and, at times, long-lasting falls in blood pressure may result from as little as 12.5 mg. of hexamethonium in some cases, in others doses of 200–300 mg. may have little or no effect.

Should excessive bleeding occur during the administration of methonium,

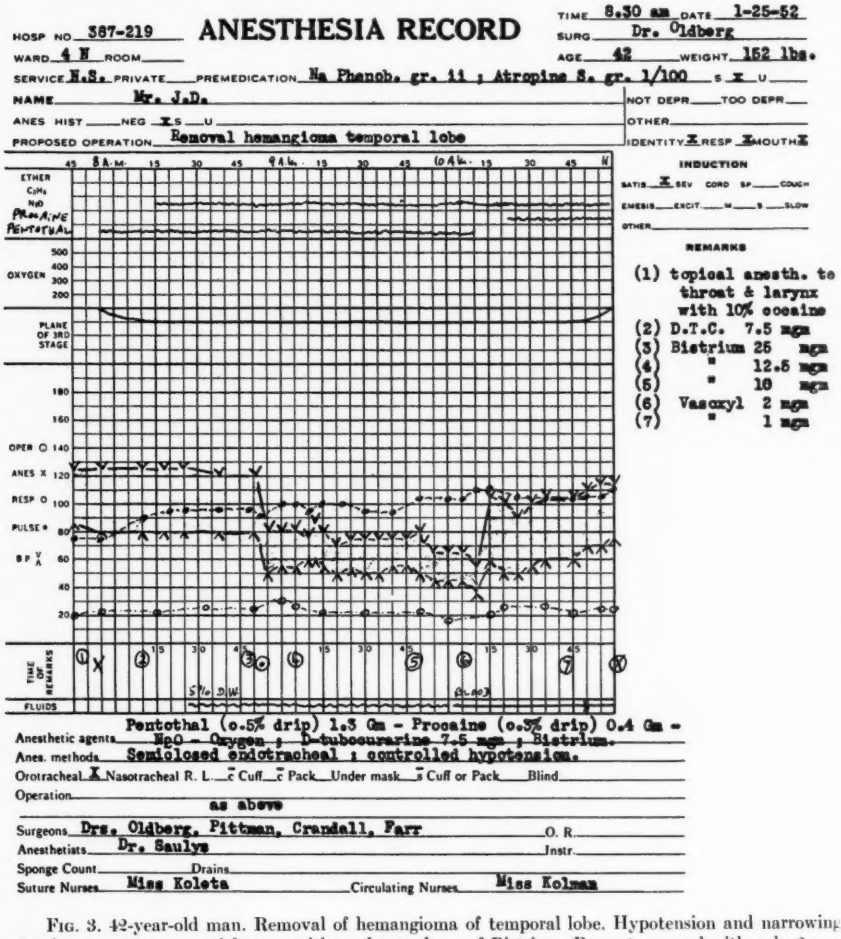
HOSP NO. 387-219
ANESTHESIA RECORD
TIME 8.30 am DATE 1-25-52
SURG Dr. Oldberg
WARD 4 W ROOM
AGE 42 WEIGHT 152 lbs.
SERVICE N.S. PRIVATE PREMEDICATION Na Phenob. gr. ii ; Atropine S. gr. 1/100 S X U
NAME Mr. J.D.
NOT DEPR TOO DEPR
ANES HIST NEG X S U
OTHER
PROPOSED OPERATION Removal hemangioma temporal lobe
IDENTITY X RESP X MOUTH X
INDUCTION
SATIS X SEV CORD SP COUGH
EMESIS EXCIT M S SLOW
OTHER
REMARKS
(1) topical anesth. to throat & larynx with 10% cocaine
(2) D.T.C. 7.5 mgm
(3) Bistrium 25 mgm
(4) " 12.5 mgm
(5) " 10 mgm
(6) Vasoxyl 2 mgm
(7) " 1 mgm
ETHER
Cyclo
N2O
PENTOTHAL
OXYGEN
PLANE OF 3RD STAGE
OPER O
ANES X
RESP O
PULSE •
BP V A
TIME OF REMARKS
FLUIDS
5% D/W
Pentothal (0.5% drip) 1.3 Gm - Procaine (0.3% drip) 0.4 Gm -
Anesthetic agents N2O - Oxygen ; D-tubocurarine 7.5 mgm ; Bistrium.
Anes. methods Semiclosed endotracheal ; controlled hypotension.
Orotracheal X Nasotracheal R. L. C Cuff C Pack Under mask C Cuff or Pack Blind
Operation as above
Surgeons Drs. Oldberg, Pittman, Crandall, Farr O. R.
Anesthetists Dr. Saulys Instr.
Sponge Count Drains
Suture Nurses Miss Koleta Circulating Nurses Miss Kolman

FIG. 3. 42-year-old man. Removal of hemangioma of temporal lobe. Hypotension and narrowing of pulse pressure were satisfactory with moderate doses of Bistrium. Prompt reversal with only 2 mg. Vasoxyl.

prompt replacement therapy is imperative, as the vasomotor system is no longer under control. Blood volume must be maintained assiduously lest shock supervene. Needless to say, oxygenation must be adequate at all times, and capillary refill-time prompt. The skin should be warm and pink, denoting good capillary circulation, the superficial veins somewhat dilated—respiration slow—and in all a pattern of a normal physiologic state otherwise.

The pulse rate usually varies but little if excessive hypotension is avoided. In younger individuals there may be a moderate increase in pulse rate, but bradycardia is seen rarely.

Hexamethonium (C6) [Bistrium®] has largely replaced pentamethonium (C5) as it is a more potent blocking agent, and is said to have more parasympathetic blocking powers than C5. It is noteworthy, however, that at times one will give a satisfactory response where the other has failed.

Hypotension induced with the methonium compounds can be reversed promptly by the use of vasopressors, preferably those acting peripherally, such as norepinephrine (Levophed®) and phenylephrine (Neosynephrine®).[9] Methoxamine (Vasoxyl®), however, is useful too. A small dose of these drugs may cause an exaggerated shift of blood pressure; 2–5 mg. of methoxamine generally are adequate, but may be repeated if needed. Hemostasis can be assured if the method of narrowed pulse pressure is employed. Thus reactionary hemorrhage is an insignificant problem. Yet, the blood pressure can be restored to normal readily before operative closure or should the problem at hand at the moment demand it.

It still remains a matter of speculation whether the combination of a methonium compound and an adrenal blocking agent or adrenalytic compound such as Dibenamine is the answer in cases which prove resistant to methoniums alone. To date, we have not put this to a practical test. We have, however, found that control of hypotension is markedly improved if Procaine is infused simultaneously.

This method of controlled hypotension is not a simple one. It requires skill in administration and, above all, sound judgment for its use. It should not be used in arteriosclerotic individuals, nor should it be entrusted to the novice. Slight indiscretions in the conduct of hypotensive anesthesia may mean the difference of life or death to the patient. Above all, it must be indicated definitely by the problems at hand, rather than to save a bottle of blood for transfusion. It must never be employed after severe bleeding has been encountered and the patient is consequently in a state of incipient shock.

We feel that indications for the use of this technique are operations for aneurysms, vascular tumors of the brain and kidney, or upon major blood vessels. We do not feel, however, that this technique is justified in the average operative case. It has inherent dangers which must constantly be borne in mind, regardless of the method by which hypotension is induced. The methonium drugs, we feel, are not the ultimate choice, but we do believe that they are a step in the proper direction toward controlled hypotension.

SUMMARY

Controlled hypotension and the various techniques to achieve it are discussed.

The authors prefer the pharmacological blocking agents, particularly hexamethonium (Bistrium®) to the other methods available, despite the fact that this drug is somewhat unpredictable in its action.

The indications for the use of this technique are outlined and should be adhered to. The contraindications to the use of hypotensive anesthesia are discussed, and the potential hazards of the technique are emphasized. Success and safety can be expected from it only in experienced hands.

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PNEUMOENCEPHALOGRAPHY IN CHROMOPHOBE ADENOMAS OF THE HYPOPHYSIS

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THE value of pneumoencephalography is generally conceded to lie in the assistance it gives the surgeon in planning his approach and in making possible a more nearly accurate prognosis. This diagnostic procedure, however, is seldom performed routinely in cases of pituitary adenomas. Once the diagnosis of an adenoma is established by the presence of an enlarged sella and visual and endocrine symptoms, little attention is given to determining the exact position and extension of the neoplasm, despite the fact that in some cases there is a marked discrepancy between the clinical signs and the true size and extension of the adenoma.

The size to which a neglected chromophobe adenoma may grow is illustrated by the case reported by White and Warren.[18] This man, who had lived in China, had noticed increasing loss of temporal vision and endocrine changes over a period of 10 years, but still maintained 20/40 vision on the nasal half of his left eye. He entered the Naval hospital in Chelsea following the onset of headaches, personality change, and seizures. Death followed an attempt to remove his huge chromophobe adenoma, which was found at post mortem to weigh 150 gm. and to extend from the crista galli to the pons, and upward nearly to the corpus callosum.

Large series of operations for pituitary adenomas clearly show that the low operative mortality rate of intrasellar tumors increases considerably if the adenoma extends beyond its original boundaries. Thus, the mortality rate in Jefferson's[11] series increased from 2 per cent to 33 per cent, and in the 300 operations of Olivecrona from 6.4 per cent to 35 per cent (Bakay[1]). It was pointed out in a previous paper[1] that even slight extension above the sella impairs the surgical results. When the adenoma protruded largely into the 3rd ventricle, the surgical mortality rate reached a peak of 74 per cent. But fatalities were more frequent even in patients with lesser hypothalamic involvement than in those without it, and the late results of the operation were not favorable. Patients with chromophobe adenomas are often treated by endocrinologists and internists and when the choice of surgery or X-ray treatment comes up for discussion, the surgeon usually has to give an estimate of the surgical hazard involved. Here, knowledge of the extent of the adenoma helps considerably. Sometimes there is no way to determine the size of the neoplasm except by encephalography.

TECHNIQUE

Pneumoencephalography is performed under local anesthesia through a lumbar puncture, with the patient in a sitting position. Mere visualization of the basal cisterns alone without seeing the outlines of the ventricular system is not sufficient. It is essential to visualize the ventricles, especially the 3rd, to determine the relationship between an extrasellar extension of the adenoma and the anterior portion of the 3rd ventricle. On the other hand, air in the subarachnoid spaces of the convexity of the brain should be avoided; it does not serve any useful purpose, only increasing the patient's discomfort and sometimes interfering with the interpretation of the picture by overshadowing the sellar and suprasellar structures in the lateral projection.

To obtain satisfactory filling with as little air as possible, the patient should sit fairly erect, with his head flexed forward to 45°. The head rests against the obliquely placed X-ray head unit. This amount of flexion corresponds to a 30° inclination of the orbitomeatal line which—according to Robertson[13]—favors exclusive filling of the ventricles. Five cc. of air are usually injected through the lumbar needle before withdrawing any of the CSF, except for a few drops to verify the position of the needle. Lindgren[12] pointed out that satisfactory filling of the ventricular system is more surely obtained when air is injected prior to the evacuation of fluid, although this opinion is not shared by Robertson.[14] Following the initial injection of air, CSF is exchanged with air slowly and in amounts of 5 cc. at a time. Films taken during this procedure reveal the location of air in the intracranial cavity; often the patient himself can give valuable information in this matter. Absence of pain or headache means usually that the air is passing into the ventricles. Complaints of headache and nausea indicate that the ventricular system and great cistern are filled and the surplus air is rising to the convexity of the brain or that the air is entering the subarachnoid spaces instead of the ventricles. The amount needed to outline the ventricular system varies with the capacity of these structures. As an average, 20 cc. are sufficient.

When the ventricles are filled, the head is flexed slightly backwards to obtain a maximal filling of the basal subarachnoid spaces. In this position of the head the subsequently injected air (usually 10 cc.) fills the chiasmatic and interpeduncular cisterns through the cisternae pontis. Actually, the effect of this extension of the head is twofold: (1) it creates a good position to fill the perisellar spaces with a small amount of air; (2) according to Robertson[13] it also serves to pass air from the dorsal part of the cisterna magna into its ventral part dorsal to the medulla, whence it rises into the ventricles.

After the air filling is completed, the lumbar needle is removed and the patient is placed upon the X-ray table in a face-up position and without turning his head around. The sitting patient simply reclines and is slipped over to the table. To facilitate this step, one uses for the air filling a dental chair, with removable back, which can be elevated to the level of the table. In our experience, a considerable part of the air trapped in the basal cisterns can escape toward the convexity if the patient is turned around. This is also the reason for taking the crucial films immediately after the patient has been placed in the face-up position. This series of films, of course, is completed by taking the lateral and P-A views, after the patient has been turned on his abdomen; however the most important ones are taken first. These include the A-P and brow-up lateral view, preferably stereoscopic ones. These pictures include the usual views with brow up (the orbitomeatal line is

vertical) and with the central ray directed perpendicularly to the sagittal plane of the head. In addition, lateral views are also taken with the head overextended. For this purpose the patient lies supine, the head hanging over the edge of the table and resting with its vertex on the head unit, which is lowered accordingly. The degree of overextension varies from patient to patient, depending mostly on the length and adiposity of the neck. Moderate tilting backwards of the head is sufficient, and it is by no means necessary to get the orbitomeatal line in horizontal position. Sometimes better pictures are obtained with the usual brow-up position, although pictures taken with the head in overextension carry the additional advantage of outlining better the anterior part of the 3rd ventricle, especially if it contains only a small

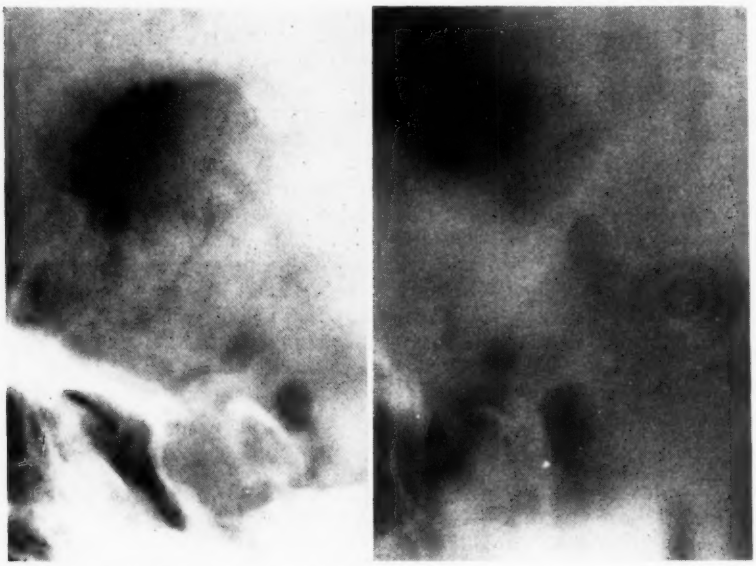

FIG. 1. Pt. A.C. (*Left*) Encephalogram of a pituitary adenoma with slight suprasellar extension. (*Right*) Midline laminagram, performed at the same time, outlines the 3rd ventricle.

amount of air. Nevertheless, it occasionally happens that the 3rd ventricle cannot be outlined in the lateral pictures, although the presence of air in it is demonstrated on the A-P view. However, in lateral projection this thin layer of air can be overshadowed by air in the temporal horns and cerebral sulci. In such cases laminagraphy may be used. A lateral laminagram, aimed at the structures of the midline, clearly outlines the "hidden" 3rd ventricle as well as the sagittal cross section of the sella and the basal cisterns (Fig. 1). This usually requires a central lateral section as well as sections of 0.5–1.0 cm. to both sides of the midline.

RESULTS

The present series is based on the pneumoencephalographic findings of 14 chromophobe adenomas with extrasellar extension. Seven of the cases are from the Neurosurgical Service of the Massachusetts General Hospital and

7 are from the Neurosurgical Service of the Serafimer Hospital (Stockholm, Sweden). These adenomas can be classified in 3 different groups on an anatomical basis, according to the size and shape of their suprasellar portion. This is not only a simple method of classification, but has also some practical implications as far as treatment is concerned.

Type 1. These adenomas are characterized by a massive extension of the neoplasm, directed upwards and sometimes backwards. There were 6 such cases in our series. They show a greatly enlarged, shallow sella with marked destruction, and with decalcification, and frequently almost complete absence of the posterior clinoid processes. Air filling of the basal cisterns reveals a massive protrusion of the adenoma in the cranial direction, which reaches 2–3 cm. above the normal level of the diaphragma sellae.

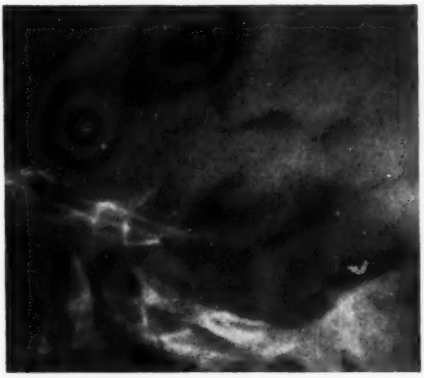

FIG. 2. Pt. H.L. Large pituitary adenoma with huge suprasellar extension. Pneumoencephalogram shows the chiasmatic and interpeduncular cisterns being elevated and spread apart.

The clinical symptoms varied considerably from case to case. One would expect these patients to be rather neglected ones, with symptoms of long standing and with marked visual deterioration and pituitary insufficiency. Often, however, this is not true. In 2 patients (both 26 years old), clinical signs did not date back longer than 3–10 months; one of them had no impairment of sexual potency and the other did not suffer from menstrual irregularities. All of these patients had a more or less complete bitemporal hemianopia, but 3 of them had remarkably good vision and very little evidence of optic atrophy. It is important to bear this in mind when one compares the clinical signs with the encephalographic findings.

As is to be expected from adenomas of such size, the basal cisterns and even the anterior portion of the ventricular system underwent profound change. The lateral pneumoencephalograms characteristically show the chiasmatic and interpeduncular cisterns, which under normal circumstances converge upwards, spread apart (Fig. 2). Under the pressure of the tumor mass, which grows between them, both cisterns take a vertical position. Besides the change in position, these cisterns are also deformed, and the anterior and medial portion of the interpeduncular cistern is as a rule distorted or obliterated. Rarely are they completely obliterated. The cisterna chiasmatis was absent in 1 instance only; in another it contained but very little air. In the rest of the patients this cistern, as well as the interpeduncular one, was quite large. The interpeduncular cistern was present in all cases and

seemed to be diminished in 2 patients only. This fact might be explained by the slow growth of the neoplasm and the absence of increased intracranial pressure, which gives the neighbouring structures time to adapt themselves to the new situation. Other extracerebral tumors also wedge themselves into the subarachnoid space, and as the top of the tumor indents the surface of the brain, a more or less marked dilatation of the subarachnoid space occurs, especially around the base of the tumor.[4] A similar mechanism keeps the basal cisterns patent around the extending adenoma. Variations are considerable as the size of these cisterns differs even under normal circumstances.[15]

The 3rd ventricle was deformed in all of these cases when the upper end of the tumor bulged into the floor of the anterior portion of the ventricle; lateral encephalograms revealed characteristic convex filling defects. In 2 of the patients the entire 3rd ventricle was pushed backwards.

In half of the cases the anterior horns of the lateral ventricles were deformed by the tumor mass protruding into their floor.

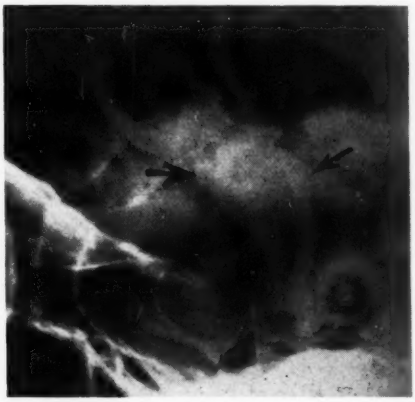

FIG. 3. Pt. K.K. Chromophobe adenoma with massive extrasellar extension of the "hourglass" type. The superior portion of the adenoma protrudes into the 3rd ventricle up to the foramen of Monro.

Type 2. This type, represented by 2 cases, does not differ from the previous type as far as the clinical signs are concerned. The sella in these patients was only slightly or moderately enlarged, without much destruction; the posterior clinoids were present although decalcified. One would have expected the adenomas to be small, but the encephalograms showed the true size of these lesions. They extended upwards far into the middle cerebral fossa; the suprasellar extensions were larger than the intrasellar portion of the adenoma. These neoplasms were of a curious shape, the large extrasellar portion being connected with the smaller intrasellar one with a rather thin neck, just like an hourglass tumor of the spinal canal (Fig. 3). Consequently, the chiasmatic and interpeduncular cisterns, which were located around the connection between the two portions of the tumor, contained a fair amount of air.

Both anterior horns of the lateral ventricles revealed an impression in their floor, but the most affected part of the ventricular system was the 3rd ventricle. The suprasellar portion of the adenoma, situated just anterior to the 3rd ventricle, bulged into it in anterior-posterior direction, in one of the cases just below the foramen of Monro.

Tumors of this type represent a difficult surgical problem, owing to their compression of the hypothalamus. The role of pneumoencephalography in

these cases is to reveal the gravity of the situation before surgery is undertaken. Without knowing how formidable the extension is, the surgeon might even overlook the upper portion of the adenoma.

Type 3. This group of cases is characterized by a small sella containing a small intrasellar adenoma with a smaller or larger suprasellar extension directed, in needle-like fashion, upward and usually backward. Four of the cases were of this variety. In only 1 was the sella enlarged beyond its physio-

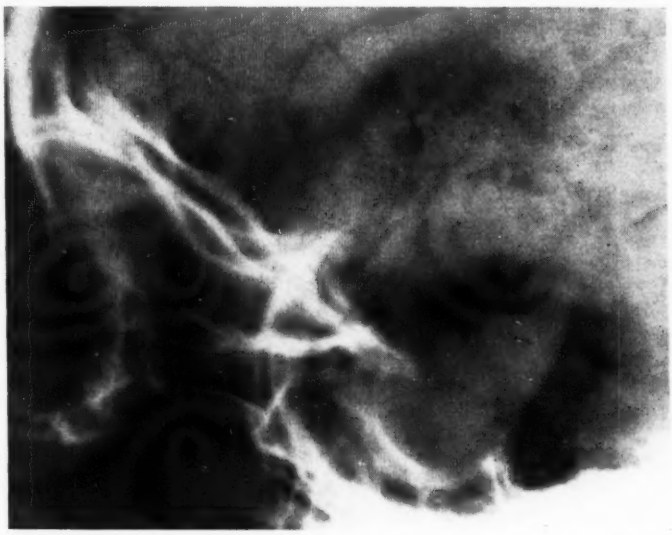

FIG. 4. PT. A.F. A cystic adenoma, most of which is situated above the sella. There is a large amount of air in the basal cisterns. The sella is slightly enlarged.

logical limits; in the other 3 it was not definitely enlarged, although there was some atrophy of the posterior clinoids in all but 1, which in X-ray configuration was perfectly normal.

These patients represent real diagnostic problems in spite of the fact that their symptoms—both visual and endocrine—are usually typical. The normal size of the sella is most misleading, yet the series reported by Davidoff and Epstein[5] reveals that such cases are by no means rare. We have had patients referred for investigation with the following comment: "Normal size of sella should exclude pituitary tumor." Another patient with a slow onset of temporal hemianopia and early menopause was treated as a case of retrobulbar neuritis for 6 years! Even when the clinical diagnosis of chromophobe adenoma is inevitable, the difficulty of the situation is very often minimized by assuming that the adenoma is small. None of these patients showed signs of marked panhypopituitarism. On the other hand, there is almost always in such cases an expansion of the adenoma in the upward

direction and the danger of rapid visual impairment can be great. Pneumoencephalography should be performed to determine not only the diagnosis, but also the type of treatment.

On encephalograms the extent of the extrasellar portion can be measured. In 1 case the pituitary mass protruded only slightly above the confines of the expanded sella, and the laminagram showed no encroachment of the 3rd ventricle. In the rest of these patients, however, the extension was more pronounced—up to 2–2.5 cm. above the diaphragma sellae—and the discrepancy between the size of the sella and the tumor was marked (Fig. 4). The width of the base of these suprasellar extensions is not great, compared to their vertical extent; while they elevate the hypothalamus and bulge into the anterior part of the 3rd ventricle from below, they do not obliterate the basal cisterns. The chiasmatic and interpeduncular cisterns are spread apart, but always contain sizable amounts of air, and the air of the interpeduncular cistern frequently circles around the extension on both sides, showing on stereoscopic films the forceps-phenomenon of Davidoff and Epstein.[5] The anterior horns of the lateral ventricles were normal in all cases.

To demonstrate the difficulty involved in dealing with the treatment in these cases, abbreviated case histories of 3 of the patients of Group 3 are presented below.

Case 1. MGH 656402. F.B., a 52-year-old female, was admitted on Aug. 3, 1950. For the previous 9 years she had slowly and progressively lost the vision of her right eye and had had occasional bitemporal headaches. A hysterectomy had been performed in 1938. For 2 years there had been deterioration of the left eye also. Fifteen months prior to admission she had been examined in another department of the hospital, and at that time there was a complete temporal field defect on the left side; on the right side only the upper nasal quadrant was present. Marked right optic atrophy was found. X-rays of the sella were reported normal and on that basis, observation was decided upon.

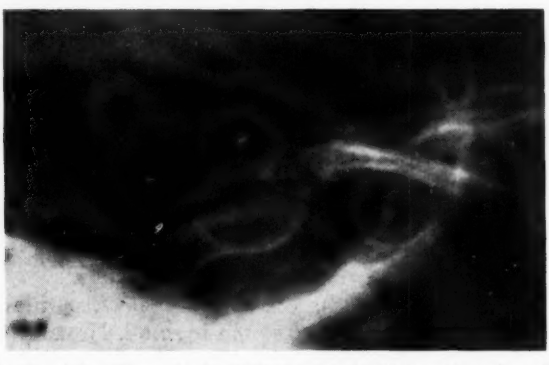

FIG. 5. Pt. F.B. The size of the sella turcica is within physiological limits. Air outlines the adenoma which protrudes out of the sella upwards and backwards.

Examination. On admission to the Neurosurgical Service, the patient's right eye was blind; the left showed temporal hemianopia. X-ray films of the skull outlined a normal-sized sella with somewhat atrophic posterior clinoids. Pneumoencephalography revealed a small suprasellar mass, presumably adenoma (Fig. 5).

Operation (by Dr. W. H. Sweet). The sella was exposed from a right transfrontal approach. The left optic nerve seemed to be in fairly good condition, but the nerve on the right crossed over a tumor mass which occupied all the area beneath the chiasm. The neoplasm also extended into the right parasellar region, encroaching on the right internal carotid artery. Its removal could therefore not be carried out completely.

Diagnosis. Adenoma of mixed type, predominantly chromophobic.

Course. Intensive postoperative X-ray treatment was given. The patient has been doing well up to the present time, but there was no improvement in her vision after operation.

Comment. A coincidence of several factors led to almost complete blindness in this patient before any attempt was made to treat her. Her previous hysterectomy deprived later investigators of a cardinal sign of chromophobe adenomas—the cessation of menstruation. Her normal sella was undoubtedly responsible for a prolonged period of "observation"; chromophobe adenoma was suspected but thought to be small. What was responsible for her symptoms proved to be a rather small suprasellar extension, which probably could have been removed years ago.

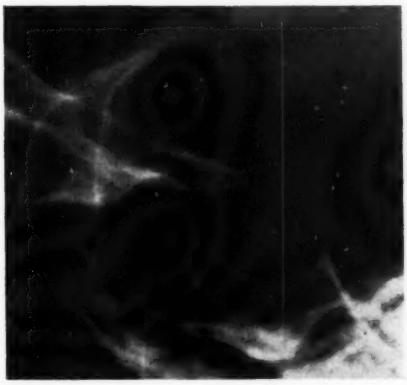

FIG. 6. Pt. A.F. The sella is not enlarged but the floor appears slightly ballooned and the posterior clinoids are thinned.

Case 2. MGH 729511. A.F., a 41-year-old female, was admitted on May 27, 1952. Her menopause had begun 7 years previously. Shortly before that, her last child was born and ever since she had been having secretion from her breasts. In the last 3 years she had gained weight and had also had hazy vision. Bitemporal hemianopia had been observed over the last 18 months, developing first in the left and then in the right eye. She complained also of dull frontotemporal headaches. Fifteen months previously she had been admitted on another service of this hospital, and the diagnosis of a chromophobe adenoma was made. At that time clear-cut bitemporal hemianopia was found, with temporal pallor of both discs. Vision of the right eye was good, but it was 20/200 in the left eye. The sella was not enlarged (Fig. 6), but the posterior clinoids were thinned and the floor of the sella appeared to be slightly ballooned. There were no signs of panhypopituitarism. Radiation was decided upon and during the next 2 months a 3500 r tumor dose was delivered to the sella through both lateral, intraocular and occipital portals, using the 2-million supravoltage machine. During the irradiation there was a transitory impairment of the left visual field involving the upper nasal quadrant.

Examination. When admitted to the Neurosurgical Service, the patient complained of headaches. Her vision had deteriorated; the right eye was unchanged, but she had become blind on the left. There was no change in her endocrine status, except for the secretion of her breasts, which had ceased. There was some progression in the thickening and enlargement of her sella. Pneumoencephalography revealed a suprasellar mass continuous with the soft tissue in the sella turcica and measuring about 2.2 cm. in height and 1.6 cm. in width (Fig. 4).

Operation (by Dr. J. C. White). Right frontal craniotomy was done and a large bluish-red tumor, reaching about 2 cm. above the level of the anterior clinoid processes and compressing the left optic nerve, was removed. This mass contained 10 cc. of brownish-red fluid.

Diagnosis. Chromophobe adenoma, partly degenerated.

Course. The patient recovered satisfactorily. However, up until the present time her visual fields have remained unchanged.

Comment. When first seen, this patient had no signs of either imminent blindness or pituitary insufficiency. The sella was almost normal. A small adenoma, but no suprasellar extension was suspected. It is likely that during the X-ray treatment central necrosis developed within the adenoma. Subsequent blindness of the left eye was not helped by surgery. If she had been referred to the Neurosurgical Service earlier, operation would have been performed immediately after demonstrating the actual extent of the lesion by pneumoencephalography. The vision of the left eye could thereby have probably been saved.

FIG. 7. Pt. M.D. Small adenoma of the hypophysis with a pinlike extension upwards, as visualized by encephalography.

Case 3. MGH 648929. M.D., a 47-year-old female, was admitted on Feb. 8, 1949. She had had a rather sudden menopause 7 years before, followed by a gain of about 30 lbs. in weight, which was lost subsequently by a reducing diet. During the last year she had lost some of her hair. About a year before admission she had first noticed a shadow across the left temporal visual field.

Examination. There was left temporal hemianopia, with loss of sight in the upper temporal quadrant on the right side. There was bilateral optic atrophy, more pronounced on the left. Roentgenograms of the skull showed thinning of the posterior clinoids and a questionable slight expansion of the sella turcica. BMR was −21 per cent. Pneumoencephalography revealed a small adenoma with a pinlike suprasellar extension (Fig. 7).

Course. During the following 5 days X-ray treatment versus surgery was considered. Even during this short period of time, however, visual acuity decreased and vision in the lower temporal quadrant of the right field was lost. Craniotomy was decided upon.

Operation (by Dr. J. C. White). The sella was exposed through the left transfrontal route. Medial to the left optic nerve was a red fleshy mass. A piece of the capsule was excised, and the suprasellar portion and part of the content of the sella were removed.

Diagnosis. Chromophobe adenoma.

Course. Recovery was uneventful. The patient's vision improved considerably; 1 year after operation she had a full right visual field.

Comment. This patient had a rather typical history of chromophobe adenoma with moderate visual symptoms. The sella was small and the adenoma was thought to be one for which radiation should be tried. A well-circumscribed suprasellar extension (demonstrated by pneumoencephalography) caused rapid deterioration of the vision of one eye; immediate surgery restored the vision.

DISCUSSION

Balado and his co-workers[2,3] pointed out as early as 1927 that the visualization of filling defects of the 3rd ventricle is of great value in the surgery of pituitary lesions. At the same time Heidrich[8,9] called attention to encephalography as an important asset to the diagnosis of intrasellar and suprasellar lesions, and pointed out the occasional discrepancy between the clinical signs and the true extension of the tumor. He also believed that this procedure could be relied upon to determine whether the intracranial or transnasal surgical approach should be attempted. Both of these investigators made their films from the lateral angle with the head tilted backwards, and with the brow up, a method which later became used routinely by Lysholm and Twining to visualize the anterior portion of the 3rd ventricle.

Twining, in his fundamental paper[17] on the radiology of the 3rd and 4th ventricles, presented a case of acromegaly with a huge eosinophilic adenoma of the hypophysis bulging into the 3rd ventricle. He stated that the clinical diagnosis is usually very easy and air studies are not often indicated. In this particular case, however, the patient's vision was so bad that there were

doubts as to the size of the tumor, and the purpose of ventriculography was to discover how high the tumor reached. Nevertheless, air studies were used with some reluctance and applied in no more than 8 per cent of their pituitary tumor material. Pneumoencephalography in cases of pituitary neoplasm was used more frequently by Lysholm and Lindgren in Stockholm.

The first report on the use of laminagraphy to visualize the 3rd ventricle was that of Twining.[16] Its use combined with encephalography in the diagnosis of midline tumors (including 1 pituitary adenoma) was described by Epstein and Davidoff.[7]

Deformities of the chiasmatic and interpeduncular cisterns as shown in encephalograms of pituitary adenomas were described by Dyke and Davidoff[6] in 1934. Later, Davidoff and Epstein[5] presented the results of 34 encephalograms of pituitary adenomas. There were 3 cases in this series in which the sella turcica was of normal size, and a considerable number of additional cases have been encountered since the termination of their statistical series. The 3rd ventricle was normal in 6 cases only. The cisterna chiasmatis was normal in 2 instances; it was obliterated in 12 cases and displaced in 7 others. This shows a higher percentage of obliteration than our series, but it also demonstrates that the chiasmatic cistern is not necessarily absent when a pituitary adenoma extends above the sella turcica. This might be of importance in differentiating between adenomas and true suprasellar tumors. According to Schlezinger and Teplick,[15] clear visualization of this cistern is sufficient practically to exclude the possibility of a suprasellar tumor. The latter, originating at the level of the cistern or above, tends to press upon it from above and obliterate it at an early stage, whereas extensions of pituitary adenomas—pushing the tuber cinereum upwards—do not occupy the chiasmatic cistern, unless they reach a large size.

Relatively small adenomas with sharply upward-directed extensions (Type 3) should be detected by pneumoencephalography and treated surgically, if possible, as they carry the potentially acute danger of visual deterioration.

It has been pointed out in several papers relating to this problem that the overwhelming majority of patients with pituitary adenomas subjected to pneumoencephalography present clinically difficult diagnostic problems. Instead of limiting its use to cases with unusual features, encephalography should be employed more widely and play a greater role in the follow-up of nonsurgically treated cases. It has been customary in many clinics to apply X-ray treatment alone, even in those cases of chromophobe adenomas that cause visual symptoms, provided these symptoms do not threaten blindness. We believe that radiation should never be undertaken without exact determination of the extent of the adenoma by pneumography. Naturally, such patients should also be observed afterwards very carefully for any deterioration of symptoms. In a great majority of cases follow-up examinations have included only testing of visual fields at regular intervals with occasional X-ray examination of the sella. We believe that in many cases

these measures alone are not satisfactory in avoiding the development of practically inoperable extensions of the adenoma. The neoplasm may grow out of the sella to a considerable extent without making the hemianopia worse. This type of extension can proceed to a critical point, finally making surgery very hazardous. It is also known, on the basis of considerable statistical data,[1,10] that a more or less typical bitemporal hemianopia is present in no more than 65 per cent of the cases. When homonymous hemianopia and other atypical forms of visual defect that indicate deviation from the midline and lateral extension of the growth are present, the operative mortality rate is increased and the results are often disappointing.[1] Adenomas that cause blindness of one eye are especially difficult to follow as no sign indicates further extension on the side of the blind eye. To follow the growth of the neoplasm by comparing the bony outlines of the sella alone can be fallacious as adenomas with a tendency to grow above the sella may cause hardly any enlargement of the sella. For these reasons we believe that repeated air filling of the cisterns surrounding the sella turcica should be included in the follow-up examinations of nonsurgically treated pituitary adenomas and that they must be used at the slightest suspicion of increased growth. Frequently it is the only information obtainable regarding the size of extension. Its more widespread use would help reduce the number of cases in which the patient comes to operation too late to preserve useful vision and to permit safe removal of the adenoma.

SUMMARY

A technique to fill the 3rd ventricle and the basal cisterns with small amounts of air through lumbar encephalography is described.

Changes in the form of the ventricular system and of the chiasmatic and interpeduncular cisterns caused by chromophobe adenomas with extrasellar extension are described on the basis of 14 encephalograms.

Three different types of chromophobe adenomas have been distinguished, based on the shape and size of the suprasellar extension and its relationship to the sella turcica.

Adenomas with a narrow-based upward extension associated with normal-sized sella are especially suitable for surgical removal.

The importance of pneumoencephalography in surgical planning as well as in the follow-up of nonsurgically treated adenomas is emphasized.

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THE SURGERY OF CRANIUM BIFIDUM AND SPINA BIFIDA

A FOLLOW-UP REPORT OF SIXTY-FOUR CASES

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With improvement in the surgical management of congenital defects, emphasis has been shifted from operative mortality and survival to operative salvage and restoration of function. This report is an analysis of the results in a series of 64 cases of spina bifida and cranium bifidum operated upon over the past 15 years. The series was a selected one only in the sense that surgery was usually withheld from babies with severe hydrocephalus and paralysis. In none of the cases did hydrocephalus appear or increase after surgery nor, as far as could be determined, was the neurologic defect exaggerated.

Questionnaires were sent to parents and physicians of all 64 patients; response was obtained in 51 cases; 33 patients reported for re-examination.

SPINA BIFIDA

The operative procedure employed in this series was essentially the same in all cases. The meningoceles were dissected in layers down to the vertebral canal. The myelomeningoceles were treated by replacing all the neural elements within the vertebral canal. The dural layer was trimmed and sutured. The other tissues were closed in as complete layers as possible over the dura mater. No attempt was made to correct the skeletal defect in the vertebra. When the local tissues were inadequate for tight closure of the defect, fascial flaps were mobilized from the adjacent muscles and imbricated over the defect. To permit closure in a few cases, the superficial tissues were dissected widely with the assistance of relaxing distal incisions; the distant defects so produced were closed with skin grafts.

Forty-eight patients were operated upon; there were 34 cases of myelomeningocele, 10 of meningocele, 2 of lipoma and 2 of occult spina bifida. Forty-two patients were operated upon in the first year of life and 6 after the second year. Of the 48 cases, follow-up has been obtained on 40. Three patients have died since discharge from the hospital. Two patients with hydrocephalus and myelomeningoceles died 2 months and 2 years postoperatively. The third patient, having had a meningocele repaired, appeared to be normal at discharge but died 4 months later as a result of whooping cough.

In reviewing all the cases known to the Surgeon General's Office up to 1905, Moore[6] found the operative mortality rate to be over 35 per cent after

the 5th year. This is not the case in modern series[3,5] and in our experience; children only a day or two old tolerate the procedure as well as do the older patients. The improved results probably reflect the better pediatric management available at present and the better appreciation of the problems inherent in the surgery of the newborn. The antibiotic drugs seem to have played only a small role, since approximately half of the present series antedated the availability of these agents.

Some of the complications of spina bifida, particularly bladder and gait disturbances, are difficult to evaluate until the child is over 2 years of age. For this reason we hesitate to classify a child as normal until he is past this age. Sixteen patients (Table 1) in the spina bifida group, who have been followed for 2 to 15½ years, appear to have no neurologic defects. Three now appearing to be normal have been followed for only 12 to 15 months.

TABLE 1

*Results following operations for spina bifida**

	Total Cases	Cervical	Thoracic	Lumbar	Lumbo-sacral	Sacral
Myelomeningocele	34	I-*1*-①	II-*1*-①	III-*6*-②	III-*5*-①	III-*4*
Meningocele	10	II-*1*-①	I	I ①	II ①	
Spina bifida occulta	2			I	*1*	
Lipoma	2				I-*1*	
Total	48	7	5	14	15	7

* Roman numerals indicate no neurologic deficits; italic numerals indicate neurologic deficits; encircled numerals indicate status unknown.

Eighteen of the patients (Table 2) who were followed for 2 years or more were found to have some degree of neurologic deficit. In 13 cases this consisted of motor disturbances, atrophy and weakness. Five are unable to walk, but 1 is ambulatory with braces and crutches. Another, only 28 months old, can walk with assistance, so further improvement seems likely.

Twelve patients show some type of sensory loss. Two of these, having cervical lesions, show only proprioceptive loss in one upper extremity.

TABLE 2

*Types of neurologic deficits after operation for spina bifida**

18 Patients	Myelomeningocele	Meningocele	Spina Bifida Occulta	Lipoma	Total
Motor disturbance (atrophy and weakness)	10	1	1	1	13
Unable to walk	5				5
Able to walk	4	1		1	6
Unknown	1		1		2
Sensory loss	9	1	1	1	12
Sphincteric disturbance	10		1	1	12

* Two of the patients listed with deficits in Table 1 are not included in this table, having died before reaching the age for proper evaluation.

Urinary incontinence has been the most distressing postoperative sequel. Usually this is not definitely discernible until the patient is more than 2 years of age. Twelve patients suffer this complication. Two have no other neurologic defect; 6 of them also suffer from some degree of weakness or sensory loss which does not interfere with normal activities.

In 3 cases, surgical attempts have been made to correct the urinary incontinence. In one, the ureters were implanted into the colon but, because of poor rectal sphincter control, the situation has not been improved. In 2 cases, urologists have attempted to create functional external sphincters by fascial-sling procedure suggested by Cooney and Horton.[1] These children have not been observed long enough to determine the utility of the procedure. This operation can be done only upon males so, even if effective in that sex, incontinence remains the most formidable barrier to rehabilitation of these children.

Of the 48 patients with spina bifida, 8 (17 per cent) displayed associated congenital anomalies. These consisted of 5 cases of club feet, 1 Klippel-Feil malformation with multiple skeletal abnormalities, 1 Arnold-Chiari malformation, and 1 case of congenital hip dislocation. Two patients had hydrocephalus at the time of operation and both have died. A third baby, suspected of being hydrocephalic at the time of surgery when 3 months old, is completely normal 9½ years later with no evidence of an enlarged head.

CRANIUM BIFIDUM

Sixteen patients with cranium bifidum were operated upon and follow-up was obtained in 11 instances. The defects were all in the occipital area, 12 being classified as encephaloceles, 3 as meningoceles and 1 as a lipoma involving the central nervous system and its coverings. One patient died 3 weeks postoperatively and another died 6 months after surgery. A third child, with mental retardation and hydrocephalus, died 3 years postoperatively. Three children are normal and have been followed for 6½ to 11 years. Two others appear normal but have been followed less than 2 years. Two patients show marked mental retardation and a third has strabismus, impaired visual acuity and a head-nodding type of tremor.

In this group the incidence of associated congenital anomalies was higher than in the spina bifida cases, there being one or more anomalies in 6 of the 16 cases (36.5 per cent). These included 1 case of cleft palate, hare lip and hydrocephalus, 1 congenital defect of the parietal bone, 1 congenital absence of the occipital plate and cerebellar vermis with a Klippel-Feil malformation, 1 case of hypospadias, 1 Arnold-Chiari malformation, and 1 instance of agenesis of the right lung in a baby with a Klippel-Feil deformity.

COMMENT

The management of the anomalies, cranium bifidum and spina bifida, can not be exclusively a neurosurgical problem. The assistance of experienced pediatricians is invaluable in the immediate pre- and postoperative periods.

The close collaboration of other specialties—particularly orthopedics, urology and rehabilitation teams—is needed as the child develops.

Great strides have been made in aiding these patients to overcome muscular disabilities but the problem of urinary and rectal incontinence has remained unsolved. Fisher, Uihlein and Keith[3] in a follow-up study found vesical disturbances in 76 of 148 patients with myelomeningoceles and 3 of 5 cases of spinal meningocele. Coughlin[2] reported 5 instances of sphincteric paralysis in 12 patients, none of whom were improved by surgery.

The age at which these patients were operated upon does not seem to be an important factor in regard to operative mortality or prognosis.

Hydrocephalus should not be a deterrent to repairing the defect if an adequate amount of cerebrum remains. Some of the patients may need one of the various fluid-shunting procedures, but repair of the defect does not seem to initiate or accelerate the hydrocephalus.[2,3,4,5]

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EXPERIENCES WITH HYPOPHYSECTOMY IN MAN*

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THE present investigation concerns the effect of removal of the pituitary gland in some conditions in which it was thought probable that hypophysectomy might favorably influence the future course of the primary disease.

From a clinical point of view there are three important questions connected with hypophysectomy in man: the rationale of the operation, our possibilities of judging of the completeness of the operation, and the pre- and postoperative treatment of the patient. These points will be discussed briefly, and a more detailed report will be given of the surgical procedure.

Hypophysectomy was performed in the following conditions:

1) *Cushing's syndrome*, when there was no improvement after irradiation of the pituitary region, or if after a period of improvement the patient had become resistant to further irradiation.

2) *Malignant hypertension*—severe, with grade 4 eyeground changes. Sympathectomy in these cases rarely gives even temporary relief. An intact pituitary-adrenal system is a prerequisite for the genesis of experimental essential hypertension, and hypophysectomy eliminates the condition. In clinical cases of hypertension the blood pressure has been lowered after damage to the pituitary or the adrenals. Administration of DCA to these patients made the hypertension reappear.

3) *Diabetes mellitus*—severe, with diabetic retinopathy and chronic renal disease, when proper treatment with an adequate dose of insulin did not stop the progress of the vascular changes. It has been suggested that this type of diabetes might involve the hypophysis-adrenal cortex to a greater degree.

4) *Carcinomas of the breast and of the prostate gland*. These tumors sometimes react favorably to changes in the hormonal milieu brought about by castration, treatment with certain hormones, and—as has been shown lately—by adrenalectomy. The effect of the hormone treatment in these cases has been ascribed to a depressing action on the hormone production of the pituitary.

We also had in mind some other malignant tumors as possibly being responsive to the change in the hormonal milieu brought about by hypophysectomy: chorionepitheliomas and malignant melanomas.

* Given as a Mayo Foundation Lecture at the Mayo Foundation, Rochester, Minnesota, on November 20, 1952.

Our first results were published in March 1952.[4] In 1936 Elden[2] reported a case in which the pituitary gland had been destroyed by electrocoagulation by Van Wagenen. Part of the pituitary gland was still present at the autopsy.[3] The patient had presented the clinical picture of Simmonds' disease. Chabanier *et al.*[1] in 1936 reported on hypophysectomy in a case of severe diabetes mellitus. After removal of the pituitary gland the diabetes could be reasonably well controlled. There was no autopsy in this case. From the fact that the patient after operation required as much as 70–80 units of insulin per day, we are inclined to believe that functioning pituitary tissue was still present. In 1952 Perrault[5] briefly reported on a case of carcinoma of the breast with pulmonary metastases, in which hypophysectomy had been performed 2 months earlier by Le Beau. This resulted in improvement of the general condition of the patient and increase in weight. The fact that the adrenalin test was normal in our opinion makes it probable that the hypophysectomy was incomplete. In 1952 Naffziger[6] and collaborators reported the result of hypophysectomy in a case of malignant melanoma. The patient died 2 months after operation from progress of the malignant condition. There was still a minimal rest of pituitary cells lining the dural covering of the pituitary fossa.

SURGICAL PROCEDURE

The hypophysis is surrounded by a rich system of blood vessels. It is imbedded in a large dural sinus (Fig. 1), which forms a ring in the periphery of the diaphragm of the sella. Also, the dural lining of the sella is highly vascularized. These veins are not particularly large but communicate freely with the cavernous sinus on both sides, and a tear in any of these veins is apt to lead to profuse hemorrhage. Moreover, the lateral walls of the pituitary fossa are for a part identical with the lining of the cavernous sinus, which

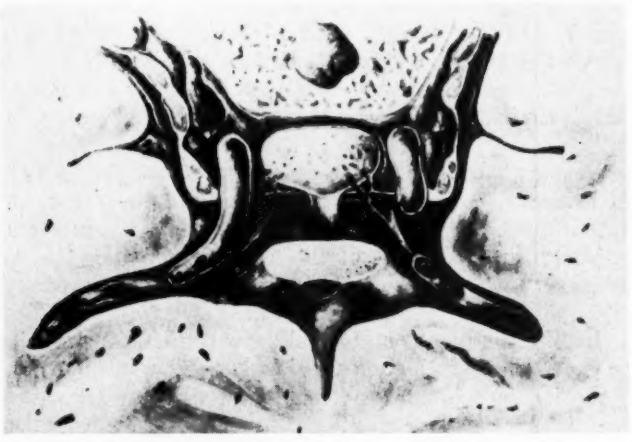


FIG. 1. Section of pituitary gland in situ showing relation to adjacent blood vessels.

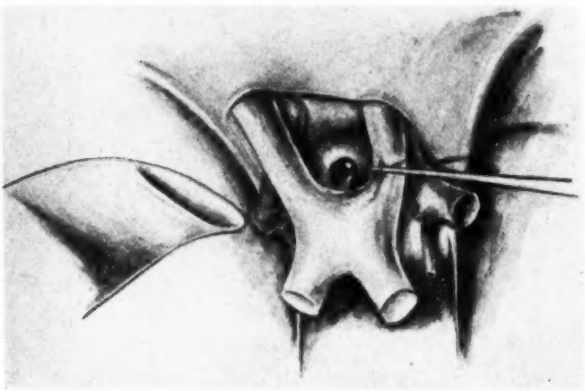


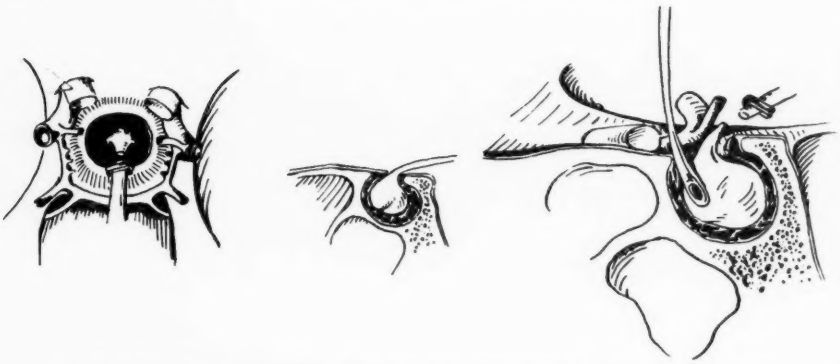
FIG. 2. Exposure of pituitary stalk.

in many cases is very thin. In addition the pituitary gland receives small arterial twigs, but these as a rule do not give much trouble except in hypertensive cases.

In our first patient, a woman with Cushing's syndrome, we tried to destroy the hypophysis by electrocoagulation with an electrode, insulated up to 5 mm. from its tip. We coagulated in as many places as possible. As will be shown later the gland was far from completely destroyed. It would be dangerous to insert the electrode close to the cavernous sinus, and in subsequent cases we therefore resorted to extirpation of the gland.

The pituitary region is usually exposed by a rather small right pituitary flap, and the arachnoid in the chiasmal cistern is removed as completely as possible. If the patient is blind in one eye the flap is made on that side, as division of the optic nerve greatly facilitates further manipulations. With intact chiasm and optic nerves the pituitary stalk is hidden beneath the right optic nerve. The nerve is retracted with a blunt hook sufficiently to expose the stalk, which, when seen from the side and above, appears to run in an almost horizontal direction (Fig. 2). When the chiasm is prefixed and the anterior margin of the sella is overhanging, it may be difficult or impossible to expose the stalk. It may then be necessary to split the chiasm in the midline to get sufficient access to the diaphragm of the sella and the stalk. Since a really prefixed chiasm is rare, we have found it possible in nearly every case to expose the pituitary stalk without doing anything to the optic nerve or chiasm.

After the stalk has been exposed, it is clipped and divided (Figs. 3 and 4). Usually the yellow glandular tissue of the pituitary can be observed through the diaphragm, which is thin around the insertion of the stalk. In the periphery a venous ring of varying size can be seen, and the diaphragm is opened just inside this ring. A circular incision of the diaphragm is completed with a small blunt hook and, if necessary, some cutting current. The gland is mobilized with blunt instruments. In favorable cases the entire gland can be lifted up from the sella and removed. Quite often, how-



FIGS. 3–4. The pituitary stalk is divided, and the gland is mobilized by blunt dissection.

ever, troublesome bleeding occurs in mobilizing the gland, necessitating piecemeal removal with rongeurs and spoon.

Even when the gland has been removed apparently intact and hemorrhage controlled, permitting inspection of the empty sella, curetting often brings out small pieces of glandular tissue adherent to the lateral and anterior walls. We have found it quite difficult to remove the gland completely. Even if a tiny piece of viable glandular tissue is left behind, it may regenerate, and the result will be vitiated. So far, the best method of removing pituitary remnants is to pack the sella with a cotton pledget, and then inject it with Zenker's solution. This is then allowed to remain in the sella for a couple of minutes. The procedure is repeated two or three times, until the lining of the sella appears thoroughly cauterized.

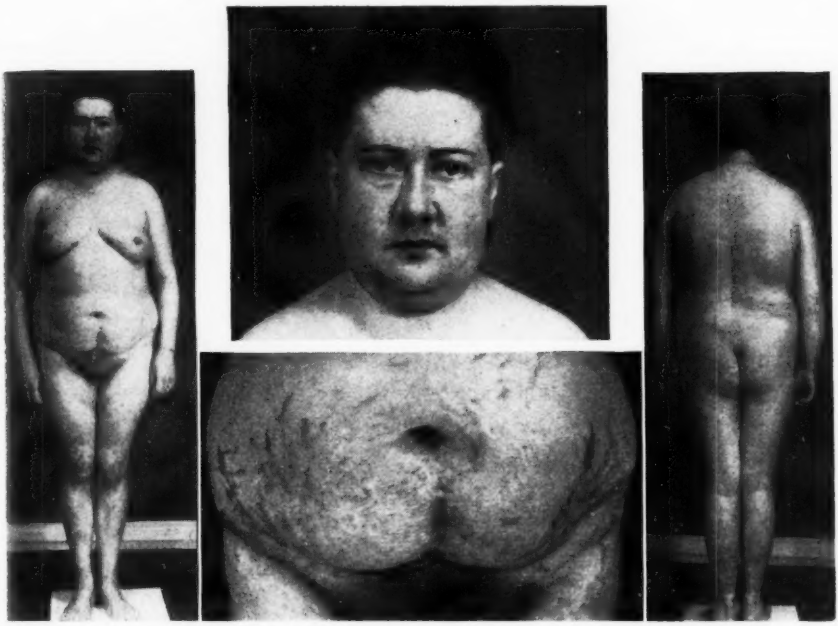
Unless the sella is completely dry, inspection and cauterization of its dural lining is impossible. If hemorrhage makes a thorough inspection impossible, the sella is packed with a piece of gel foam, and the wound is reopened a few days later. After removal of the gel foam the above-mentioned procedures can be carried out without difficulty. Operation in two sessions was necessary in a number of our cases.

Up to October 15, 1952, we had performed hypophysectomy in 26 cases. There were 3 cases of Cushing's syndrome, 7 of malignant hypertension, and 4 of diabetes. The remaining 12 were cases of malignant tumors.

CASE REPORTS

CUSHING'S SYNDROME

Our 1st case of Cushing's syndrome was that of a girl, born in 1927, who had had typical symptoms since the age of 15. She had never menstruated, but normal menstruation appeared and all the symptoms disappeared after irradiation of the hypophysis in 1943. Improvement lasted until 1948, when she was given another series of x-ray treatment. This was without effect, and so still another series was given in 1950. At examination in June 1951 she presented an advanced stage of Cushing's syndrome: she was bedridden because of her asthenia, had the typical ap-



FIGS. 5–8. Patient with Cushing's syndrome before coagulation of the pituitary gland.

pearance (Figs. 5–8), a B.P. of 200/120 mm. Hg and amenorrhea of several years' duration.

On June 26 and July 6, 1951 the pituitary gland was thoroughly coagulated in the manner described above. There was little reaction to the operation.

Postoperatively there was a very rapid improvement: in a few months her appearance was completely normal, the B.P. had returned to 120/80, and menstruations reappeared. At present, in October 1952, she looks and feels well and has even become pregnant (Figs. 9 and 10).

The 2nd case, in which the pituitary gland was also coagulated, is of less interest, since the patient died a few days after operation from suffocation caused by an undiagnosed bronchial carcinoma.

In the 3rd case, a 19-year-old girl had presented Cushing's syndrome for 2 years. Two months before admission she became psychotic and completely negativistic, did not eat or drink, and it was felt that x-ray treatment would act too slowly to save her life.

She was therefore operated upon on Aug. 19, 1952. At hypophysectomy care was taken to leave a small piece of gland.

At first there was very little reaction to the operation, but on the 2nd day the systolic B.P. suddenly fell to 50. It rose on noradrenaline. She then did fairly well, but on the 3rd day became restless and cyanotic, the B.P. fell, and she died within a few minutes.

At autopsy nothing was found that could explain her death.

FIGS. 9–10. Cushing's syndrome (same patient as in Figs. 5–8) 1 year after coagulation of the pituitary gland.

DIABETES MELLITUS

Hypophysectomy for diabetes was performed in 4 cases, in 3 of them because of advanced changes in the eyegrounds and renal disorder, and in 1 because of beginning retinal disease and resistance to insulin as well as frequent attacks of coma.

The 1st patient, a man of 30, had had diabetes since the age of 2. His diabetes was controlled with 80 units of insulin per day. Since 1950 progressive hemorrhages in the eyegrounds had occurred and led to blindness of both eyes. At the same time hypertension, 180/100 mm. Hg, appeared and there were signs of progressive renal damage.

Hypophysectomy was performed on Nov. 30, 1951.

The patient made an uneventful recovery, and later tests indicated that the hypophysectomy was complete. The BMR dropped to −20; there was no drop in the number of circulating eosinophil cells with adrenalin and ACTH. The B.P. dropped to 125/80, and renal function improved with disappearance of the albuminuria. His diabetes is now controlled with 12 units of insulin per day, which completely removes the glycosuria. The blood sugar remains at about 150–200 mg. per cent on this insulin dose.

In the following 3 cases the outcome was unfavorable.

In the 2nd case, a young woman of 20 had had diabetes since the age of 6. She had spent most of the last 3 years in hospitals, as slight irregularities would bring about a precomatose condition. Her daily insulin dose was about 200 units.

Hypophysectomy was performed on May 19, 1952.

There was no immediate reaction, and her insulin dose could be reduced to about 0 units on the next day. She continued to do well until May 21, when she became comatose. Her blood sugar remained at 200; there was no acidosis.

Since the presence of a clot could not be excluded, the flap was reelevated on May 22. There was considerable edema of the brain, necessitating frontal lobe resection, and she died on the same day.

The pituitary had been completely removed save a small rest, which was largely necrotic but contained a layer of viable cells.

The 3rd case was that of a man of 28 who had had diabetes since the age of 11. He had diabetic retinal disease with blindness of the left eye and beginning impairment of vision of the right eye. There was moderate impairment of renal function.

Hypophysectomy was done on May 5, 1952.

On the evening of the same day the patient became comatose with Cheyne-Stokes respiration. The flap was immediately reelevated, but nothing was found except for swelling of the brain. He died a few hours later.

At autopsy only a slight swelling of the left hemisphere was observed. The pituitary had been completely removed save a small rest, which was largely necrotic but contained a small number of viable cells.

The 4th patient was a young woman, aged 24, who had had diabetes since the age of 10. She had severe diabetic retinal disease and cataracts with reduced vision of both eyes, and a B.P. of 160/110 with moderate impairment of renal function.

Hypophysectomy was performed on June 10, 1952.

There was very little immediate reaction. The insulin dose could soon be reduced from 60 to about 8 units a day, and her B.P. went down to 105/75. Her recovery was uneventful except for slight infection of the soft tissues and possibly of the bone flap. This caused occasional elevation of the temperature and some edema of the right eyelid, which, however, responded well to chloromycetin. She continued to do well but suddenly on July 11 she became subcomatose and later had epileptic fits. On July 12 status epilepticus occurred, and the patient died.

Autopsy showed nothing that could explain her epileptic fits and sudden death. Serial sections of the pituitary fossa showed that the gland had been completely removed. We had thought, in view of the evidence of infection of the soft parts during early convalescence, that meningitis was the cause of death. However, lumbar puncture done when the epileptic fits first occurred showed only 46 cells, and microscopically the brain and meninges were normal.

MALIGNANT HYPERTENSION

Seven patients with malignant hypertension were operated on. They had eyeground changes of grade 3–4. Cerebral vascular accidents with hemiplegia or hemiparesis had occurred in 5. They all had impairment of renal function, which in some cases was severe. The average age was 32, the oldest patient being 43 years old. Signs of myocardial damage were present in all. These cases thus formed a group of young people in an advanced stage of

malignant hypertension with a presumably very short life expectancy.

The results of hypophysectomy in this group were disappointing. The 2 patients who stood the operation best and lived longest both were found at autopsy to have a chronic glomerulonephritis. When blood pressure began to drop, a week or two after operation, uremia appeared, and DCA had to be given in addition to cortisone to raise the pressure again. Nevertheless, both patients finally succumbed to their uremia, 2 and 5 months after operation respectively.

The remaining 5 cases were classified as essential hypertension. One of these patients died from the effects of an extradural clot and 1 from an acute subdural hematoma. The remaining 3 all died from acute cerebral edema. The day after operation the patients became drowsy, had signs of a contralateral hemiparesis and became comatose. Reelevation of the flap showed no hematoma but considerable edema of the brain and increased intracranial pressure. One of the patients recovered after the tip of the frontal lobe had been resected to permit closure, and did well for almost a month. She then had another similar attack and died within 12 hours. Autopsy showed considerable swelling of the brain.

In the last case of hypertension in which operation was performed, the pituitary stalk was divided under local anesthesia but nothing was done to the gland. There was a marked drop in blood pressure. The next day the patient was drowsy and had a slight left-sided hemiparesis. On the 3rd day he became deeply comatose, and reoperation was performed. There was considerable pressure from swelling of the brain but no clot. Death followed a few hours later.

The occurrence of acute cerebral edema after hypophysectomy (or division of the stalk) appears to be peculiar to endocrine disorders such as diabetes and Cushing's syndrome and also to essential hypertension in its malignant form. This reaction does not occur, at least not in this violent form, after hypophysectomy in cases of malignant tumors. A moderate degree of cerebral edema with drowsiness and even a subcomatose condition on the 2nd or 3rd day after operation was occasionally observed in the tumor cases, but never of a degree sufficient to indicate reelevation of the flap. These patients invariably recovered after a short time. The profound disturbance of cerebral function after hypophysectomy in endocrine disorders and malignant hypertension, therefore, in some way appears to be linked to the condition for which operation was performed.

MALIGNANT TUMORS

Hypophysectomy was performed in 12 cases of malignant tumors, all in an advanced stage of the disease, after all other available methods of treatment had been tried and had failed. There were 1 case of carcinoma of the prostate gland, 1 of malignant chorionepithelioma, 1 of hypernephroma and 9 cases of carcinoma of the breast. The operation was well tolerated in every case; there were no deaths or serious complications of any kind.

Our single case of *cancer of the prostate* was that of a man aged 68 who had had

signs of the disease since 1946. He had been treated by electroresection of the prostate in 1946, and in 1950 by orchidectomy followed by implantation of stilbestrol. After the end of 1951 there was steady progression of the disease, with severe pain and difficulty in urination and defecation. No signs of bone metastases were found.

Hypophysectomy was done in two sessions on April 8 and 22, 1952. There was a marked improvement. The pain disappeared within 2 weeks, and he could urinate without difficulty. Tests indicated that hypophysectomy was incomplete. Difficulties of urination and defecation reappeared after about 3 months, while the relief of pain lasted until his death on Aug. 4, 1952.

Autopsy showed that the pelvic organs were enclosed in a cancerous mass with extensive metastases to the lymph glands along the aorta. The adrenals were small (total weight 8.5 gm.), and serial sections of the pituitary fossa revealed small rests of regenerating glandular tissue (Fig. 11).

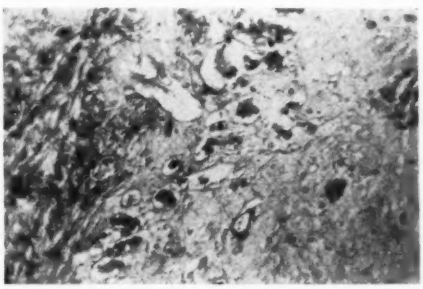


FIG. 11. Section of the pituitary fossa 3½ months after hypophysectomy for prostatic cancer. Small rests of pituitary tissue are still present.

In a case of *hypernephroma* with numerous metastases to the lung, hypophysectomy was well tolerated. However, the adrenalin test indicated that removal was probably not complete. It has not been possible to reexamine the patient, as she lives in another country. The latest report received 3 months after operation indicated progression of the disease.

The case of *malignant chorionepithelioma* was that of a girl aged 18 who had had an abortion in October 1951. In January 1952 she had an acute abdominal hemorrhage from perforation of the uterus caused by a malignant chorionepithelioma. The uterus was completely removed. Recovery was uneventful.

Early in March 1952 she was readmitted to the surgical clinic with severe pains in the back radiating down the legs. An incomplete paraplegia with excruciating pain and difficulty in urination and defecation developed within the next few weeks. Roentgenograms of the lungs showed two small patches, which had the typical appearance of metastatic tumors. Myelograms showed an extradural tumor at the level of the 12th thoracic vertebra. The excretion of chorionic gonadotrophic hormone was up to 70,000 units per day, even though the uterus had been removed 2 months previously. The diagnosis, therefore, was chorionepithelioma with extradural metastases and metastases to the lungs.

Hypophysectomy was performed in two sessions, on April 24, and May 6, 1952. Tests indicated that the gland had been completely removed.

She improved rapidly, and her pains disappeared. She was even able to walk with support. The excretion of chorionic gonadotrophic hormone dropped from 70,000 units per day to 480 units in the middle of June, and to between 13 and 53 units by September. At the end of September the tests for the excretion of this hormone were negative. X-ray of the lungs now did not show any metastases. Her condition, however, became somewhat worse, with increasing pains and difficulty of urination. Myelograms indicated that the extradural tumor was still present.

On Oct. 9, 1952, laminectomy was performed, with disclosure of an extradural mass to the left and in front of the spinal cord. The tumor was excessively vascular. The hemorrhage was impossible to control, and the patient died from exsanguination in spite of numerous transfusions.

Autopsy revealed the surprising finding that the extradural lesion was not a chorionepithelioma but an arteriovenous aneurysm emptying by large veins into the common iliac vein. There were no signs of chorionepithelioma anywhere in the body. The hypophysis had been completely removed (serial sectioning of the sella turcica).

Our interpretation is that the removal of the uterus with its attending ligatures of pelvic veins activated the arteriovenous aneurysm by obliterating some of the draining channels. After hypophysectomy the reduction of blood volume and blood pressure probably reduced the tension in the aneurysm with some relief of pressure on the spinal cord. That chorionepitheliomatous tissue was present in the body at least up to April was shown quite conclusively by the excretion of chorionic gonadotrophic hormone, which after hypophysectomy gradually dropped and ceased completely about 5 months later. It is far less probable that the metastatic tumors might have disappeared spontaneously after extirpation of the uterus, because of the excretion of 70,000 units of gonadotrophic hormone 4 months after removal of the primary tumor.

The 9 cases of *carcinoma of the breast* were all very advanced. These patients usually had huge recurrences after previous mastectomies and in most cases pulmonary and other metastatic lesions were present. Five of the patients were operated on quite recently, 2 after first having had cerebellar metastatic tumors removed. They all did well after hypophysectomy, but

FIG. 12. Patient with carcinoma of the breast immediately before hypophysectomy. Note the large number of subcutaneous metastatic tumors.

FIG. 13. Cancer of the breast (same patient as in Fig. 12) 5 weeks, 4 months and 8 months after hypophysectomy.

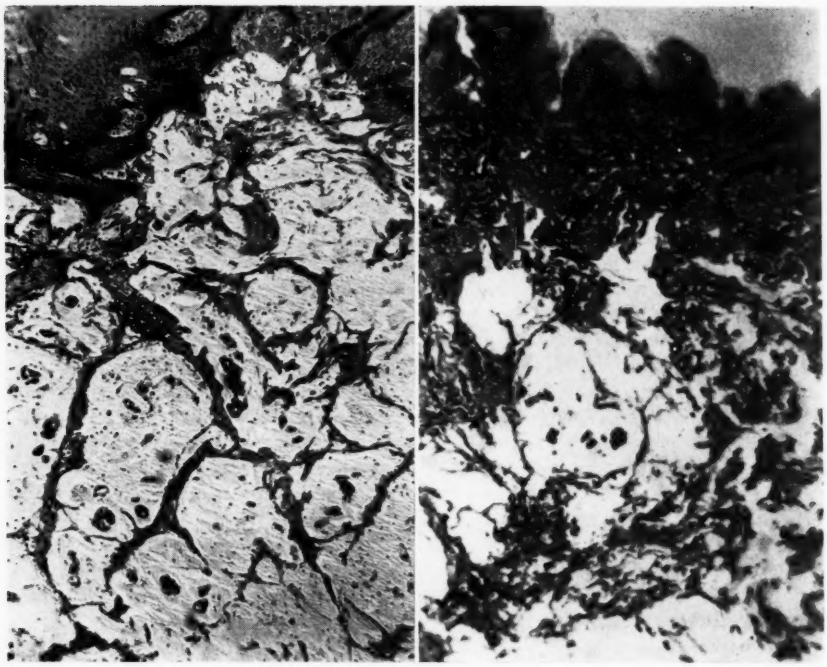
it is still too early to decide if the removal has been complete, and how the carcinomas are going to react. In 2 earlier cases the adrenalin test indicated that functioning pituitary tissue was still present, and these patients showed no appreciable improvement. They are both alive, 8 and 5 months, respectively, after operation. In a third case it was never possible to make an adrenalin test. Her carcinoma did not show any improvement, and she died 3 months after operation.

One of the patients with carcinoma of the breast was a woman aged 49. In 1945 the left breast was removed. She did well until 1948, when recurrence in the skin was observed. X-ray treatment was given with good effect; in addition she was castrated by irradiation. Later in 1948 new metastatic tumors appeared in the skin; treatment with testosterone was now given with some temporary effect. In September 1949 a cancer of the right breast developed. Treatment with radioactive phosphorus had some temporary effect, followed by a rather rapid progression. At examination in January 1952 her general condition was comparatively good, considering the enormous ulcerating cancer (Fig. 12). No bone or pulmonary metastases were found.

Hypophysectomy was performed on Jan. 29, 1952. There was very little immediate reaction, except for frequent rises of temperature probably caused by resorption from the large ulcerating cancer.

About a month after operation small islands of epithelium appeared here and there on the ulcerating surface. After that there was a more rapid improvement, as may be seen in Fig. 13. Eight months after operation the entire ulceration was covered with epithelium, and the subcutaneous cancer encasing the thorax had largely disappeared. Biopsies made at intervals of 2–3 months from January to September 1952 showed a striking change in the histological appearance of the tumor

FIG. 14. Biopsy of carcinoma of breast before hypophysectomy (patient shown in Figs. 12 and 13). Adenocarcinoma with tendency to gelatinous degeneration and signet-ring formation.



FIGS. 15 and 16. Same case as in Fig. 14. (*Left*) 4 months after hypophysectomy. (*Right*) 9 months after hypophysectomy. Only isolated carcinomatous cells are now visible.

(Figs. 14–16). The general condition of the patient has improved. She is feeling well and is able to do all her housework. All tests indicate that hypophysectomy was complete.

Our experiences with hypophysectomy in cases of carcinoma of the breast are too recent to permit any conclusion concerning the effect of the operation upon the cancer. They show, however, that hypophysectomy is well tolerated, and that reactions similar to those observed in endocrine disorders and malignant hypertension do not occur. They also indicate, though more evidence is needed here, that unless hypophysectomy is complete only a temporary effect of short duration is to be expected. On the other hand, when the hypophysectomy is complete, as in the case just reported, a remarkable regression of the carcinoma may occur. Of course, we do not know how long this improvement is going to last. It is reasonable to expect that sooner or later the carcinoma will resume its progression.

TESTS OF PITUITARY FUNCTION

As to the judgment of the completeness of the hypophysectomy, we have primarily made use of earlier well known tests of the function of the hypoph-

TABLE 1

Laboratory tests before and after hypophysectomy in a case of cancer of the prostate (Case 1) and a case of severe diabetes mellitus (Case 2)

		Case 1			Case 2	
		Before Op.	After 1 Month	After 3–4 Months	Before Op.	After 1 Month
Adrenalin test; eosinophils	Before	—	140	210	760	943
	After 4 hrs.	—	70	46	360	860
ACTH test; eosinophils	Before	—		327	500	1260
	After 4 hrs.	—		283	100	1100
Water load test	Excretion	600	495	585	635	
	Dilut.	1.006	1.006	1.006	1.002	
	Conc.	1.022	1.009	1.007	1.024	
17-keto mg./day		2.4	4.5	4.2	4.2–7.7	0.6–1.0
BMR		—	—	−5%	−3%	−27%
I^{131}	1. day excretion			46.7%		
	2. day excretion			13.2%	—	—
	3. day excretion			2.9%		
Insulin sensitivity				+++	0	+++
Serum cholesterol mg. per cent			220–250	150–200	274–324	216–240
Serum phosphorus mg. per cent		—	2.5	2.0–2.3	3.5–3.7	3.6–4.4
Blood pressure mm. Hg		150/105	100/75	100/75	160/110	100/70
Gonadotropin excretion MU./day		—	—	<6.5	—	<6.5

ysis, adrenal cortex, thyroid and gonads. So far, none of the tests known can give us *early* and conclusive evidence of the completeness of the operation. Later on endocrine deficiencies develop that can easily be observed.

In Table 1 are collected a number of tests performed before and after hypophysectomy in the one case of cancer of the prostate (Case 1) and in one of the cases of diabetes mellitus (Case 2). Autopsy was performed in both cases. In Case 1 the operation was followed by relief of all symptoms for about 3 months. When symptoms then reappeared the patient died within a short period of time. Minor strands of viable pituitary cells were found at serial sectioning of the sella turcica. The adrenals were somewhat atrophied, weighing a total of 8.5 gm. The thyroid gland was of normal appearance and weighed 23 gm. In Case 2 no pituitary tissue was found at serial sectioning of the sella turcica 1 month after operation. The adrenals were normal with a total weight of 15 gm. The thyroid gland weighed 25 gm. This patient

TABLE 2

Adrenalin and ACTH tests as well as urinary excretion of 17-ketosteroids after hypophysectomy

		Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9
Adrenalin test; eosinophils	Before	210	943	120	240	173	933	136	143	407
	After 4 hrs.	46	860	160	170	83	584	117	148	289
ACTH test; eosinophils	Before	327	1206	180	190			127	—	357
	After 4 hrs.	283	1100	205	230			110	—	66
17-keto mg./day		4.2	0.6–1.0	0–0.6	3.1 (implant. TP)	1.1–3.6	1.2	0.5–0.7	0.9	3.5
Diagnosis		Cancer prost.	Diab. mell.	Chorion. epithel.	Diab. mell.	Cancer mam.	Hypernephroma	Cancer mam.	Cancer mam.	Cushing
		Pituitary left (autopsy)	No pituitary left (autopsy)	Marked improvement. No pituitary left (autopsy)	Marked improvement	No improvement	No improvement	Marked improvement	Temporary improvement	Marked improvement

had received an intravenous injection of ACTH about 3–4 hours before death, which might explain the weight of the adrenals.

In Case 1 there was a normal response to adrenalin, while in Case 2 there was no decrease in the number of circulating eosinophils 4 hours after the administration of adrenalin or ACTH. The excretion of 17-ketosteroids in Case 1 was of the same magnitude as before operation; in Case 2 it went down to almost zero. The BMR was −5 per cent after operation in Case 1 and −30 per cent in Case 2.

It thus seemed possible that the adrenalin test and the excretion of 17-ketosteroids might give us information as to whether pituitary tissue was still present after operation.

In Table 2 are collected the results of these two tests in a number of cases in which hypophysectomy was performed. We are well aware of the fact that theoretically there is no absolute justification of using the adrenalin test as a test of pituitary function. However, it is obvious from Table 2 that there was no response of the eosinophils to adrenalin in those cases in which there was improvement after the operation. In these same cases the excretion of 17-ketosteroids was extremely low. We think there are strong reasons to believe that the hypophysectomy was complete in these cases.

PREOPERATIVE AND POSTOPERATIVE TREATMENT

We have been obliged in this material to take into especial consideration the necessity of preventing brain edema of any great degree. It is well established that ACTH and cortisone as well as DCA bring about a retention of electrolytes and fluid. When using ACTH and DCA, and to a certain degree even cortisone, this retention reaches its highest values during the first days of hormone administration, and is followed by a period of increased excretion. We have tried to perform the operations during this period of increased excretion. We also have to take into consideration when hypertension or organic kidney disease is present, that ACTH as well as DCA are substances that increase the blood pressure. This holds true, but to a lesser degree, for cortisone. Furthermore, ACTH and cortisone may give rise to impairment in diabetes mellitus, while DCA does not influence carbohydrate metabolism. Eschatin,* a water extract of the adrenal cortex, may be given to all patients without causing impairment of the circulation or impairment in diabetes.

As far as possible the following schedule has been followed in the preoperative and postoperative care of the patients.

Breast cancer. For 1 week before operation, cortisone 50 mg. per day, and testosterone propionate 25 mg. per day. After operation, decreasing hormone dosages. The permanent hormone treatment can often be prescribed from the 3rd or 4th week on. Cortisone is then given in a dose of ½ tablet twice daily, later on combined with a small dose of thyroxin. We have so far omitted testosterone propionate in the permanent substitution therapy in order not to obscure the results. Thus,

* Parke, Davis & Company preparation.

testosterone propionate was not given in the cases of mammary cancer reported in this paper.

Cancer of the prostate gland. The same pre- and postoperative treatment as in breast cancer but with omission of testosterone propionate.

Diabetes mellitus. Cortisone 50 mg. per day during the week before operation, under careful control of the patient's diabetes and insulin requirement. Postoperatively the cortisone dose is kept on the same level for some days. After about 1 week we usually add 10 mg. of DCA per day, and then eliminate the cortisone after some days more. The final substitution contains DCA and, in males, testosterone propionate in implantation and a small dose of thyroxin.

Hypertension. No substitution was given preoperatively. After operation Eschatin 10 ml. every 2 hours during the 1st day; then decreasing doses. The final substitution should contain cortisone, 1 tablet per day, testosterone propionate in implantation, and a small dose of thyroxin.

All substitution therapy was omitted for at least 1 week before control examinations were started after operation and later on. It should be mentioned that the patients easily tolerated the lack of substitution therapy for the 3–4 weeks of control examinations.

CONCLUSIONS

Our experiences with hypophysectomy in man comprise 26 cases: 3 of Cushing's syndrome, 7 of malignant hypertension, 4 of diabetes mellitus and 12 of malignant tumors. In Cushing's syndrome coagulation of the pituitary or incomplete removal should be made. So far malignant hypertension seems not to be suitable for this operation, at least not until some form of premedication or some change in the technic makes it possible to avoid the ensuing acute cerebral edema. The same holds true for diabetes mellitus. The carcinomas of the breast and possibly also of the prostate, as well as chorionic epitheliomas in a stage when other methods of treatment are no longer effective, in our opinion offer a promising field for hypophysectomy. In these cases the operation is comparatively free of danger and is well tolerated.

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CASE REPORT AND TECHNICAL NOTES

BENIGN SUBTENTORIAL SUPRACOLLICULAR CYST AS A CAUSE OF OBSTRUCTIVE HYDROCEPHALUS

REPORT OF A CASE

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Cystic collections of fluid occurring above the colliculi and below the tentorium have been reported in association with obstructive hydrocephalus by Penfield,[7] Sweet,[10] Dyke,[4] Childe and McNaughton,[2] Peterson,[9] Pennybacker and Russell,[8] and Torkildsen,[12] the total of such cases reported being 13. The cause of hydrocephalus in most patients was some obstructive process either in the 3rd ventricle or aqueduct of Sylvius, more commonly the latter. In at least 5 cases the cause of obstruction to the flow of cerebrospinal fluid was a tumor in the region of the aqueduct, in 3 a stenosis of the aqueduct, in 2 a tumor in or under the 3rd ventricle, in 1 a parasagittal meningioma, and in 1 ependymitis. Of greatest interest, is the route by which fluid from the lateral or 3rd ventricles reaches the region behind the pineal gland, above the colliculi and under the tentorium. In 1 case (Case 2) of Torkildsen[12] there was a rupture of the posterior wall of the 3rd ventricle just below the pineal gland caused by long-standing excessive intraventricular pressure with escape of fluid into the space above the midbrain. In all other cases in which the site of rupture of the ventricles could be determined this occurred in the posterior portion of one or both of the lateral ventricles. Pennybacker and Russell[8] found that the rupture occurred in that part of the lateral ventricle that is thinnest normally, the posterior floor of the ventricle lying just behind the fornix and medial to the hippocampal gyrus. In all reported cases the site of rupture was clearly patent, and there was free communication between the lateral ventricles and the subtentorial cyst. In each patient in whom ventriculography was done the cyst was demonstrated as an abnormal bubble of air below the tentorium, and it was felt that the cyst in itself was not the cause of the neurological abnormalities. Pennybacker and Russell[8] suggested that it might add to the already existing obstruction by further impeding the flow of cerebrospinal fluid from the posterior fossa to the large absorptive surface of the cerebral cortex. With the exception of Cases 2 and 3 of Dyke,[4] all previously reported patients with subtentorial cysts have died either of the associated pathological condition or of some complication of surgery.

Spontaneous ruptures of other portions of the ventricular system may occur in cases of obstruction to the normal flow of cerebrospinal fluid. Sweet[10] described a patient in whom a rupture of the anterior wall of the 3rd ventricle had taken place through the lamina terminalis. Torkildsen[12] surmised in 1 case that there had been a rupture into the subarachnoid space probably through the cerebral cortex.

Hamby and Gardner[5] have reported a case unlike any other found in the literature. Their patient, a 16-year-old girl, had obstructive hydrocephalus caused by an

ependymal-lined cyst over the quadrigeminal plate. They could find no communication between this and the ventricular system either by ventriculography, injection of indigo carmine into the ventricles, or direct inspection of the cyst at operation through the posterior fossa. Evacuation of the cyst resulted in relief of all symptoms of increased intracranial pressure. They were uncertain as to the origin of the cyst, feeling that it might have been brought on by a serious fall the patient had had 8 months before operation or that it might have been developmental in origin.

CASE REPORT

NCBH #116859. P.S., an 18-month-old male child, was first admitted on Aug. 26, 1950, with the chief complaint of enlargement of the head and regression of development of 4 months' duration.

Past History. This child was born last in a family of seven children, the product of a normal pregnancy and a low forceps, hospital delivery. Birth weight was 7 lbs., 14 oz. Early development was normal. He was breast fed, with addition of solid foods as indicated and vitamins in proper dosage. At 3 months the child held up his head; at 6 months he sat alone; at 8 months he stood, and at 9 or 10 months he walked. He began to talk at the age of 16 months.

Present Illness. In April 1950, 5 months before admission, he had a severe persistent cough which was diagnosed as whooping cough, followed shortly thereafter by chicken pox. In May, the mother noticed that his head was beginning to enlarge and that he stopped trying to sit or stand alone. In June, the child was taken to a pediatrician who found the head measured 19 in. in circumference. In the following 2 months the head increased to 21 3/4 in. and the anterior fontanel was large. During this time there was occasional vomiting and the patient was listless, having no desire to sit or stand and making no progress in speech.

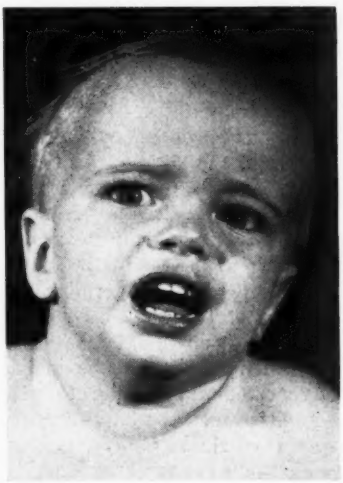

FIG. 1. Patient before head was shaved. The head is enlarged and the anterior fontanel is bulging.

Examination. He was a well developed and well nourished child with an obviously large head (Fig. 1). The head was 23 in. in circumference; chest 20 in. in circumference. The anterior fontanel was $1\frac{1}{2} \times 1$ in. and tense. There was obvious separation of the cranial sutures. There was no papilledema. Extraocular movements were normal. There was no evidence of impairment of hearing. The deep tendon reflexes were hyperactive.

Urine and blood were normal. Blood Wassermann, Kahn and tuberculin tests were negative. Roentgen films of the chest, wrist, and knee showed no abnormalities. Roentgenograms of the skull demonstrated separation of the cranial sutures with thinning of the cranial vault. The sella turcica was normal.

Course in Hospital. On Aug. 29, 1950, bilateral subdural punctures were done through the coronal sutures. No subdural fluid was obtained. The right lateral ventricle was entered at a depth of 2 cm. and clear, colorless fluid was withdrawn. A lumbar puncture was done at the same time. The cell count of both ventricular fluid and lumbar fluid was normal. Total protein of ventricular fluid was 4 mg. per cent; total protein of lumbar fluid, 22 mg. per cent.

Following this the fontanel was much softer and the child was somewhat brighter. This persisted for 4 days and it was thought that this might be a case of arrested hydrocephalus. Consequently the child was discharged to be followed at frequent intervals.

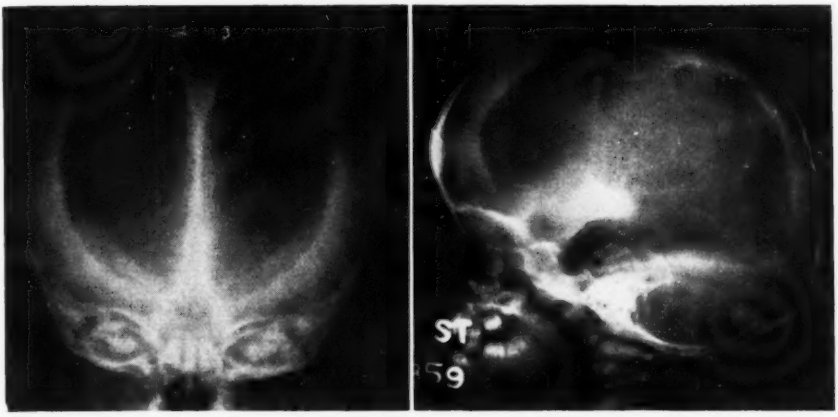

FIG. 2. A.P. and left lateral roentgenograms of skull after replacement of ventricular fluid with air. Lateral and 3rd ventricles are enlarged. There is no air in 4th ventricle or posterior fossa.

Second Admission, Oct. 10, 1950. For 2 weeks after discharge the anterior fontanel had remained soft and relaxed, but then it again became full and tense. The findings on examination were similar to those on the previous admission. The head measured 23 in.

Course in Hospital. On Oct. 11, 1950, 1 cc. of neutral P.S.P. was injected into the right lateral ventricle. After 30 minutes no dye had appeared in the lumbar subarachnoid space.

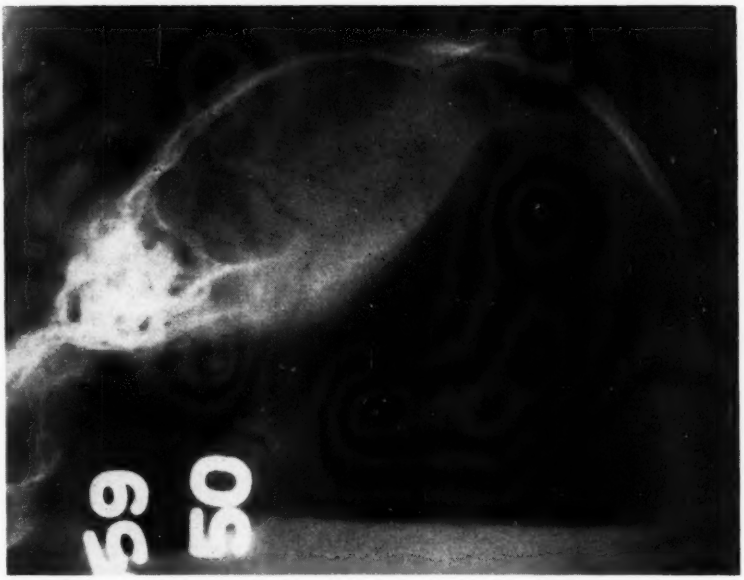

FIG. 3. Lateral roentgenogram of skull in brow-down, horizontal beam view. No air is seen below the tentorium.

It was present in the left lateral ventricle. Nine and one-half hours after injection there was still no dye in the spinal subarachnoid pathways and only a faint trace in urine collected continuously after injection of the dye.

Ventriculography. On Oct. 13, 1950 bilateral punctures of the lateral ventricles were carried out and clear fluid still containing some P.S.P. dye was removed and replaced with air. The films showed tremendously dilated lateral and 3rd ventricles (Fig. 2) and no air in the 4th ventricle. A lateral film was taken with brow down, horizontal beam. This demonstrated no abnormal collections of air and again no air in the 4th ventricle (Fig. 3).

Operation. Under endotracheal anesthesia, with the patient in the prone position, a posterior fossa exploration was done. The tension in the posterior fossa was relaxed by tapping the right lateral ventricle. The arch of the atlas was removed. When the dura mater was opened, it was apparent that the right cerebellar hemisphere was grossly enlarged. The vermis was displaced to the left side, the right cerebellar tonsil lying at the level of the axis. The left cerebellar tonsil had not herniated through the foramen magnum.

The right cerebellar hemisphere was tapped and at a depth of 3.0 cm. a cyst containing clear fluid was encountered. This hemisphere was transected and excellent visualization of the interior of the cyst was obtained. It was lined by a thin, glistening membrane, most of which was stripped away easily and without bleeding. The inferior surface of the tentorium

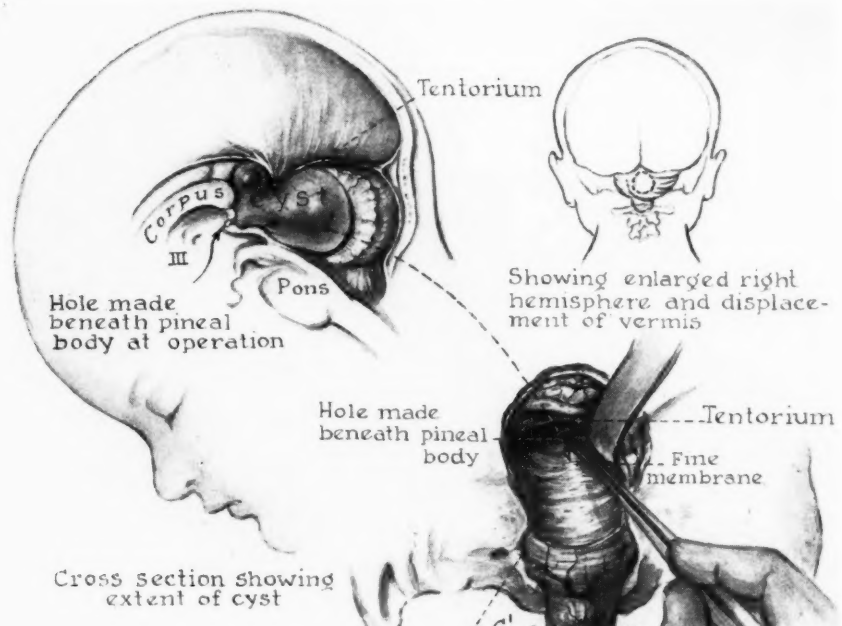

FIG. 4. Artist's drawing of the subtentorial cyst and point at which an aperture was made into the posterior 3rd ventricle.

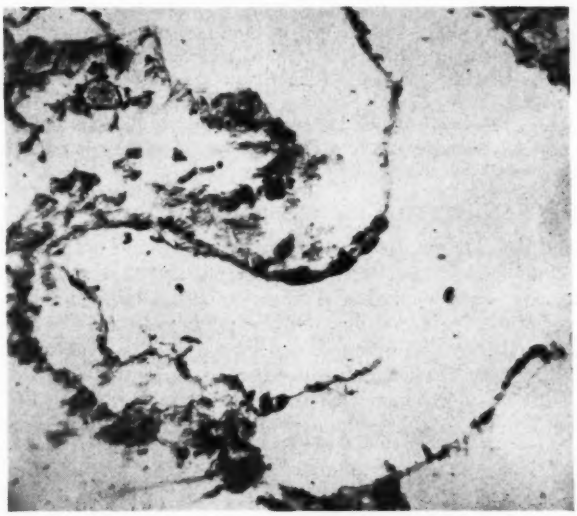

FIG. 5. Photomicrograph of lining of cyst wall. Surface cells of arachnoid are prominent, backed up by collagen fibers (×190).

was clearly visible, forming the superior surface of the cyst. Anteriorly and inferiorly were the midbrain, colliculi, and the posterior wall of the 3rd ventricle. To demonstrate patency of the aqueduct of Sylvius, a No. 8 soft rubber catheter was passed easily through the 4th ventricle and aqueduct into the 3rd ventricle for a distance of 13 cm. from the obex without meeting obstruction. Fluid aspirated from the right lateral ventricle contained 8 mg. per cent of total protein. By contrast, fluid from the subtentorial cyst contained 50 mg. per cent of total protein.

A No. 20 lumbar puncture needle was inserted into the posterior wall of the 3rd ventricle under direct visualization and fluid was aspirated. This, too, contained 8 mg. per cent of total protein. The opening into the posterior wall of the 3rd ventricle, made just below the pineal gland, was enlarged to a diameter of 1 cm. or more to provide free communication between the subtentorial cyst and the ventricular system (Fig. 4).

Postoperative Course. The patient withstood the procedure well and made a satisfactory recovery. Immediately after operation there was noticeable cavitation in the region of the anterior fontanel and this remained depressed throughout the rest of the patient's hospitalization. A P.S.P. test was repeated; dye injected into the right lateral ventricle appeared in the lumbar subarachnoid space in 11 minutes. He was discharged on Oct. 27, 1950.

Microscopic Examination (Fig. 5). Dr. Orville T. Bailey described the cyst wall as follows: "It seems to

FIG. 6. Photograph of patient Oct. 22, 1951, one year after operation.

me quite definite that the material consists entirely of arachnoid. The surface cells are very well shown and are particularly well stained in the Masson preparation. While the surface cells are rather prominent, they have the detailed structure of arachnoid cells and not of ependyma. The way that the collagen fibers are backed up against the layer of cells is also typical of arachnoid."

Subsequent Course. The patient was last seen on Dec. 22, 1951, at which time the head measured 22 in. He was showing steady improvement in his ability to talk. He was walking alone (Fig. 6).

DISCUSSION

This case and the case reported by Hamby and Gardner[5] differ from those previously described in that the cyst below the tentorium was the cause rather than the result of obstructive hydrocephalus. A ventriculogram failed to demonstrate any communication between this cyst and the ventricular system. Comparative studies of the fluid removed from the cyst and the lateral and 3rd ventricles at the same time showed a discrepancy which substantiates the contention that this cyst did not communicate with the ventricular system. The cyst in this case was lined by thin flattened cells of arachnoid rather than by ependyma as in the case of Hamby and Gardner.[5]

The fact that this cyst was lined with arachnoid cells calls to mind the cases reported by Bucy,[1] Craig,[3] Kaplan,[6] Thompson,[11] and Trowbridge and French.[13] Each of these authors described cysts in the posterior fossa, causing obstructive hydrocephalus, lined entirely with arachnoid cells. None of the cysts was in the region of the midbrain, although that removed by Thompson[11] extended into the region of the right cerebellopontine angle.

The removal of these cysts in previously reported cases resulted in complete subsidence of symptoms. Similarly the removal of most of the wall of the cyst in the case herein reported, combined with the establishment of a communication between the cyst and the posterior 3rd ventricle, has relieved the increase in intracranial pressure.

The data available on this matter favor a congenital origin for such arachnoid cysts as described here. Trauma has been considered as a cause and the increase in intracranial venous congestion associated with the coughing of pertussis may have been a causative factor. There had been no previous history suggestive of intracranial infection to favor inflammation as the cause of the arachnoid cyst. The advanced degree of internal hydrocephalus in this patient favors a long-standing obstruction, such as is often seen in congenital hydrocephalus in younger infants.

SUMMARY

1. The case of an infant with progressive hydrocephalus caused by a benign cyst below the tentorium has been reported.

2. This is the second reported case of such a cyst located at this point causing obstructive hydrocephalus. The cyst in this case was lined with arachnoid, whereas that in the case of Hamby and Gardner showed a definite ependymal lining.

3. No previously reported arachnoid cyst has been found in the supracollicular space.

4. Establishment of a communication between the cyst and the 3rd ventricle has resulted in relief of the hydrocephalus, and in the free circulation and absorption of cerebrospinal fluid.

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AN INEXPENSIVE SEMI-AUTOMATIC SERIALOGRAPHIC APPARATUS FOR ANGIOGRAPHY

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(Received for publication October 7, 1952)

Numerous articles have advocated the need for rapid serial cerebral angiography.[1–10] The apparatus required is usually expensive, or technically difficult to use. The following method of obtaining rapid multiple radiographs is presented so that the benefits of serial angiography may be had with a minimum of expense and technical personnel.

The method utilized consists of an easily constructed apparatus which allows serial radiographs to be made at one-second intervals by one technician. The x-ray cassettes are permitted to fall in position and are exposed semi-automatically through the interval timer of an x-ray machine. This is accomplished by the manual opening of the spring door, and an exposure is made when the door closes as a new cassette falls into position.

DETAILS OF CONSTRUCTION

The overall dimension of the machine is 63 inches high by 18 inches wide by 23 inches deep. This is built to accommodate a standard 10×12 inch cassette. Slight variations of width are necessary if other sizes are used (Fig. 1).

The sides are of $\frac{3}{4}$ inch plywood and are separated $13\frac{1}{4}$ inches so a 10×12 inch cassette may slide freely on its side. The front panel is in three parts. The upper and lower sections are lead-covered plywood while the middle third is masonite. The back is open except for the cross pieces of the 2×4's for stability. The plywood sides and front are built inside a framework of 2×4's for sturdiness.

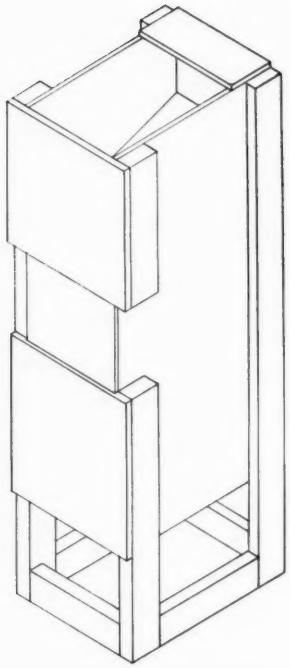
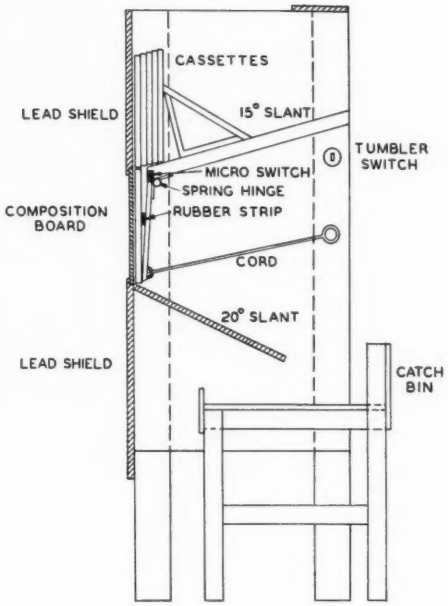
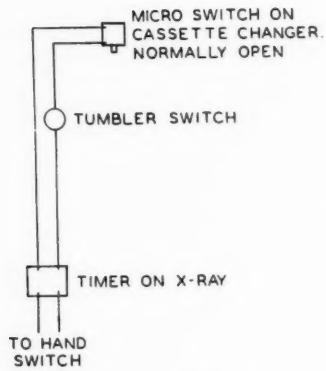


FIG. 1



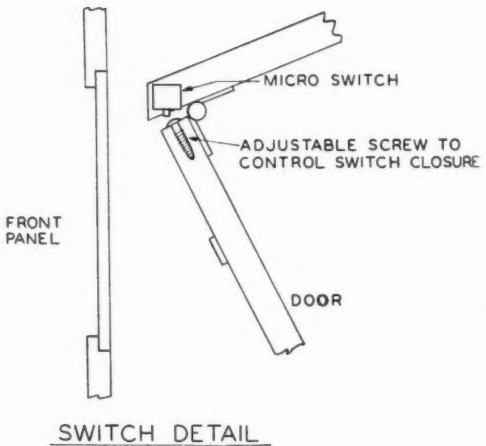
CROSS SECTION

FIG. 2



WIRING DIAGRAM

FIG. 3



SWITCH DETAIL

FIG. 4

The inside consists of two inclining planes and a spring door. The upper plane slopes at 15 degrees and is made of $1\frac{1}{2}$ inch plywood. The lower plane is a $\frac{3}{4}$ inch plywood sloping at 20 degrees, and it fits snugly to the front panel. The door of plywood is hinged by large adjustable screen-door springs to the upper plane. The front of the upper plane is separated from the front panel by slightly more than the thickness of one cassette. The microswitch (open in neutral position) is placed in the hinge side of the door, and a one inch wide strip of $\frac{1}{4}$ inch thickness of rubber is placed across the front end of the door one third of the way down. This acts as a brake to the movement of the cassettes (Fig. 2).

A small catch bin for the cassettes is placed under the lower plane. Two rubber scrub pads are all the padding required to protect the cassettes. The bin is unattached to the main apparatus to prevent jarring of the machine by the falling cassettes.

The electrical circuit (Figs. 3 and 4) utilizes a tumbler switch and a microswitch. Both switches must be closed to activate the x-ray timer. The circuit is connected to the two poles of the x-ray timer switch by means of a male-female type of plug. The closure of the two switches by-passes the hand switch of the x-ray machine.

OPERATION OF THE MACHINE

The patient lies with his back on the x-ray table and the head on a padded Mayo table. The cassettes are loaded in the machine, one directly behind the masonite, and the others on their sides in the upper inclining plane. The cassettes are held in position by the spring door and a movable wedge that slides freely in the upper plane. Under pentothal® anesthesia the internal carotid artery is injected percutaneously with 6–8 cc. 35 per cent diodrast®, and at a signal from the operator the first exposure is made by closing the toggle switch. The remainder of the exposures are made by opening the door with the pull-cord and its closure by the springs. At each door closing the microswitch activates the X-ray timer and another exposure is made.

Exposure factors are: 36 inches distance: 70 KV; $\frac{1}{10}$ to $\frac{3}{10}$ of a second; 200 MA; stationary grid. It is necessary to use the interval timer to obtain exposures in one second or less intervals for the impulse timer will not be activated properly with a short break in the contact by this electrical circuit.

Certain limitations and disadvantages are inherent in the operation of this machine. There is obviously considerable noise accompanying the closure of the spring door, but the patients are asleep and are not aware of such disturbances. Lateral views only are obtained, but these are stereoscopic views and anteroposterior views are easily taken by placing the cassette under the head. A small amount of dexterity by the technician is required, but this can easily be obtained by practice. The few disadvantages are outweighed by the simplicity of the operation, low construction cost ($20 to $25) and the adaptability provided.

SUMMARY

An inexpensive apparatus for obtaining multiple serial radiographs is presented and its construction is described. The mechanism is simple and easy to operate so that one technician may accomplish the exposures with confidence.

The author is indebted to Drs. Kenneth Livingston and Edward Davis of Portland, Oregon, for their excellent cooperation in developing and utilizing the apparatus described.

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Obituary

CLAUDE C. COLEMAN

1879–1953

Dr. Claude C. Coleman, one of the pioneers in the development of neurological surgery in this country, died in Richmond on January 9th, 1953, at the age of 73. He was born in Caroline County, Virginia on July 21st, 1879, the son of the late Frank and Jane Patrick Coleman. His early years were spent there. In 1898, after two years at William and Mary College, he taught in a country school on the Eastern Shore of Virginia for the ensuing two years and then proceeded to the Medical College of Virginia, from which he was graduated in 1903. For the next decade he pursued postgraduate training in surgery in New York and in Richmond, and during this period he was also engaged in the general practice of medicine in Buena Vista,

Virginia from 1905 to 1909. In 1913 Dr. Coleman began the practice of surgery in Richmond and soon had a wide and diversified general surgical practice. He had long had a special interest in plastic and oral surgery, and—to the end of his surgical career—he liked nothing better than to repair harelips and cleft palates.

During or just before World War I, Dr. Coleman's interests turned toward neurological surgery. He was chief of the brain surgery school at Fort Oglethorpe, Georgia at this time and later did extensive work in peripheral nerve surgery, operating in innumerable cases of nerve injuries which were chiefly casualties of the war in France. In 1919 he was associated with the late Dr. Charles H. Frazier in a military hospital in Cape May, New Jersey, and he also worked in a similar institution in Staten Island, New York.

Returning to Richmond during the latter part of 1919, Dr. Coleman began his most significant surgical achievement as organizer and director of the Department of Neurological Surgery at the Medical College of Virginia. Here he was to carry on his work as Professor of Neurological Surgery for thirty-two years until in 1951 he retired as director of the department and became Neurosurgical Consultant to the Medical College of Virginia Hospitals, a position he retained until his death. During this period, at the request of the Dean of the medical school, he also organized a department of neurological surgery at the University of Virginia in Charlottesville and served as Clinical Professor of Neurological Surgery there from 1937 to 1941, simultaneously with his work in Richmond. He thus realized his long-felt desire to establish departments of neurological surgery in the two medical schools of Virginia. In all, he held four full professorships, for in 1912 he was elected Professor of the Principles of Surgery, and in 1913, Professor of Oral Surgery and Anesthesia, both at the Medical College of Virginia. At the time of his retirement from active surgical practice in 1951, Dr. Coleman had taught a total of forty-four years in public and private schools, and in medical schools.

He was a member of most of the leading surgical and neurological societies in this country, including the Society of Neurological Surgeons, of which he was President in 1929, the American Neurological Association, the Southern Surgical Association, the American College of Surgeons, the Seaboard, Norfolk and Western, of which he had also been President, and Chesapeake and Ohio Railroad Surgeons' Associations, the Southeastern Surgical Congress, the American Medical Association, the Medical Society of Virginia, and the Richmond Academy of Medicine, of which he was President in 1925. In 1943 he was elected an honorary member of the Harvey Cushing Society.

In his later years, many well-deserved honors came to Dr. Coleman. He was appointed to the Board of Visitors of William and Mary College on April 24th, 1941 and served on that Board until 1950. He also received the Alumni Award from that school on June 7th, 1941. He was awarded the honorary degree of Sc.D. from William and Mary College in 1949. The honorary degree of Sc.D. was also conferred upon Dr. Coleman by his medical Alma Mater in June 1951. He was a member of the Founders' Group of the American Board of Surgery and served as an examiner for the American Board of Neurological Surgery from its inception in 1940 to 1949.

In World War II he served as Civilian Consultant in Neurological Surgery to the Surgeon General, and from 1946 to 1953 he was Neurosurgical Consultant to the Special Medical Advisory Group of the Veterans Administration, which met at frequent intervals in Washington, D.C.

Perhaps his happiest and proudest moment was in May 1949, when all his former

neurosurgical residents—many in prominent posts throughout the country—assembled at the Medical College of Virginia for a celebration in honor of the completion of his thirtieth year as Director of Neurological Surgery. A number of his own neurosurgical contemporaries from Boston, Baltimore, San Francisco and the Mayo Clinic also attended this meeting. In September 1951, his former and present residents presented his portrait to the Medical College of Virginia at an impressive ceremony in the Baruch Auditorium.

His medical writings were models of clarity and simple yet forceful language, as shown both in his published papers and addresses, and also in the several sections that he contributed to various volumes and systems of surgery. His achievements in the diagnosis and treatment of injuries to the head, spine and peripheral nerves were outstanding and will probably be considered in future years to have constituted his major medical contributions, together with his clarification of facial nerve lesions of all types. His excellent results in the treatment of brain abscess, brain tumor, Ménière's disease, and many other neurosurgical lesions are also well known.

To have known Dr. Coleman, even briefly, was an unforgettable, unique and rewarding experience. His sincerity and his integrity inspired loyalty and devotion in his residents and medical colleagues, and in his patients. He had an infectious sense of humor and was a raconteur of more than ordinary talent. His unique mannerisms and anecdotes of his effective methods for dealing with patients and other individuals are already both legion and legendary and are fondly and reminiscently discussed whenever his former associates, residents or internes gather together. His unusual diagnostic skill, his operative boldness or conservatism, as the individual case demanded, the general excellence of his surgical judgment, and the end results of his operations, coupled with genuine interest and keen insight in his patients' individual problems were, perhaps, his chief characteristics throughout his surgical career—a rare combination of both scientific acumen and humanitarianism. Also to be mentioned was his constant interest in his residents, not only while they were under his tutelage but afterward as well. Truly, like Osler, Dr. Coleman was the "Young Man's Friend" and he helped to further their careers whenever possible.

Dr. Coleman was married three times. In 1917 he married Julia Langhorne Cone and to them were born four children, three daughters and a son, his namesake, who is following in the footsteps of his distinguished father in pursuing a medical career. His first wife died in 1927, and in 1932 he married Ruth Threadcraft Putney, who died in 1945. In 1948 he married Constance Gooding Cardozo, who survives him, together with his four children, three stepsons and seven grandchildren.

There should finally be included in this note the tributes of Virginia's two distinguished United States Senators. At the time of Dr. Coleman's death, Senator A. Willis Robertson, his friend since early manhood when they were both practicing, the one law, the other medicine, in Buena Vista, Virginia, telegraphed the family: "Dr. Coleman's eminence in his profession was due primarily to two factors, first, an unusual and extraordinary capacity for friendship, combined with the excellent and outstanding quality of his professional work." And perhaps there could be no better eulogy than the tribute of Senator Harry F. Byrd on the occasion of the celebration in 1949 toward the close of Dr. Coleman's notable surgical career—" . . . he is the personification of all those qualities that make our nation great."

J. M. Meredith, M.D.

NOTICES

AMERICAN NEUROLOGICAL ASSOCIATION

The 78th annual meeting of the American Neurological Association will be held at the Hotel Claridge, Atlantic City, N. J. from June 15–17, 1953.

HARVEY CUSHING SOCIETY

At the 21st annual meeting of the Harvey Cushing Society, held in Hollywood, Florida on April 23–25, 1953, the following officers were elected: President-Elect, Dr. Harry Wilkins, Oklahoma City; Vice-President, Dr. Franklin Jelsma, Louisville; Secretary-Treasurer, Dr. David L. Reeves, Santa Barbara. President of the Society from 1953–1954 is Dr. Edgar A. Kahn, Ann Arbor. Members of the Executive Committee are Dr. Winchell McK. Craig, Rochester, Minn., Dr. Frank Turnbull, Vancouver, Dr. Eben Alexander, Jr., Winston-Salem, Dr. Paul C. Bucy, Chicago, and Dr. William J. German, New Haven. Mrs. William A. Nosik, Cleveland, was appointed Chairman of the Ladies Auxiliary.

NEUROSURGICAL SOCIETY OF AMERICA

The 6th annual meeting of the Neurosurgical Society of America will be held at the Broadmoor Hotel, Colorado Springs, Sept. 23–27, 1953. Attendance is for members and invited guests.

The President is Dr. William F. Meacham of Nashville, and the Secretary is Dr. C. D. Hawkes, 22 N. Manassas, Memphis, Tenn.

SCANDINAVIAN NEUROSURGICAL SOCIETY

The 9th meeting of the Scandinavian Neurosurgical Society will take place in Helsinki, Finland, Aug. 29–30, 1953. Members of American and European neurosurgical societies are cordially invited to attend. Information is obtainable from the Secretary, Dr. Olof Sjöqvist, Södersjukhuset, Stockholm, Sweden, or from Professor Aarno Snellman, Finnish Red Cross Hospital, Helsinki, Finland.

UNITED CEREBRAL PALSY

Dr. Glidden L. Brooks, of Pittsburgh, has been appointed as the first full-time Medical Director for United Cerebral Palsy, and will assume his new duties on July 1, 1953.

Dr. Charles F. McKhann, of Philadelphia, who has been active as part-time Medical Director, will continue as secretary of the volunteer Medical-Professional Executive Board of the organization and as a member of its Research Advisory Board.

In his new position, Dr. Brooks will work with the Medical-Professional Executive Board of United Cerebral Palsy, as well as the Clinical and Research Advisory Boards, and other volunteer bodies, in helping to establish standards of medical and psychological diagnosis and of clinical treatment for cerebral palsied children and adults. In addition, he will advise these Boards in granting funds for research, fellowships and scholarships, and will help organize symposia for the interchange of information about developments in the field of cerebral palsy.

INTRACAROTID INJECTION OF PAPAVERINE IN CASES OF CEREBROVASCULAR THROMBOSIS*

PRELIMINARY REPORT

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IT IS the purpose of this paper to report our clinical observations on the effect of injection of a known vasodilating agent directly into the common carotid artery in cases of thrombosis of cerebral blood vessels and ischemic cerebral infarction. This constitutes a preliminary report of a study concerned with the effect of intracarotid administration of vasodilating drugs upon the cerebral circulation in man.

The literature concerned with the treatment of patients with ischemic cerebral infarction has been replete with paradoxical statements and contradictions. Such basic considerations as the anatomic and physiologic arrangement of terminal vessels,[1,12] the occurrence or nonoccurrence of angiospasm of intracranial blood vessels,[7] the role of the autonomic nervous system in regulation of caliber of intracranial arteries and arterioles,[3,4,7] the objective results of stellate ganglion block in the treatment of cerebrovascular disease,[2,5,8] and the clinical effect of vasodilating drugs on the cerebral circulation of man[4,10] are not yet clearly understood nor has agreement among authorities been reached on any of these issues. One of the most lucid essays on this subject is that of Schmidt.[11] He says that while the possibility of cerebral angiospasm from sympathetic impulses in man can be neither affirmed nor denied, at present the preponderance of the evidence is against it. Our own clinical experience with stellate ganglion block in cases of cerebral vasoconstriction has thus far borne out this statement. Moreover, Schmidt has pointed out that the physiologic basis for failure of the commonly employed vasodilating agents to be clinically effective in increasing cerebral circulation is the apparent lesser sensitivity of intracranial vessels to vasodilator agents as compared with the sensitivity of the extracranial vessels. Hence a vasodilator agent, such as papaverine, when administered orally or parenterally in sufficient concentration to act all over the body, may bring about a redistribution of blood at the expense of the brain, plus a decrease in peripheral blood pressure and an actual decrease rather than the desired increase in cerebral blood flow.

Our investigation is ultimately concerned with several questions that bear upon the treatment of ischemic infarction of the brain. These questions are: (1) How can selective vasodilatation of the intracranial arteries and

* This study was aided by a grant from the Eli Lilly and Co., Indianapolis 6, Indiana.

arterioles in man be best accomplished? (2) Under what conditions is such vasodilatation accompanied by increased cerebral blood flow? (3) What consideration must be given to the changes in the peripheral blood pressure, cardiac rate and output, and respiratory rate which are side effects produced by certain vasodilating agents? (4) If cerebral vasodilatation and increased cerebral blood flow can be accomplished in cases of ischemic brain infarction, will it prove to be clinically beneficial, or will reactive edema, increased intracranial pressure, and later angiospasm mitigate any possible benefits?

The phase of this study with which we are concerned in this preliminary report is the effort to produce selective dilatation of intracranial vessels. The basis for attempting to accomplish this by the intracarotid injection of papaverine is simply the rationale of delivering a vasodilating agent directly into the vessels to be dilated. Mufson[9] has based his treatment of peripheral obliterative arterial disease on the same rationale. We wish to report certain of our clinical observations following the intracarotid injection of papaverine in 6 patients with complete hemiplegia caused by ischemic infarction of the brain, and in 10 additional patients in whom injection was carried out for its vasodilating effect during cerebral angiography. An evaluation of these early observations will not be attempted by us in this preliminary report, and only unequivocal objective findings will be reported.

Single and multiple intracarotid injections of papaverine, in dosage varying from 0.5 to 1.5 grains, were made in 6 patients demonstrating complete hemiplegia. In each patient the hemiplegia had been present for 72 hours or more. In each case a clinical diagnosis of cerebrovascular thrombosis with ischemic infarction of the brain had been arrived at independently by several examiners on the basis of mode of onset and development of the hemiplegia, neurologic examination, and absence of red blood cells from the cerebrospinal fluid. Each injection was made into the common carotid artery that was contralateral to the hemiplegic extremities. In 2 cases a single injection was made, in 2 cases two injections were made, and in 2 cases three injections were made. Four of the patients demonstrated an objective change in the paralyzed extremities within 60 seconds after the initial injection. Two demonstrated no observable change. Brief summaries of these clinical investigative procedures follow.

CASE REPORTS

Case 1. A.S., a 52-year-old white male, a known hypertensive, noted onset of weakness of the right extremities. This progressed to complete right hemiplegia within 24 hours. Neurologic examination revealed complete flaccid paralysis of the right extremities, sensory type aphasia, deviation of eyes to the right, and a positive response to the Babinski test on the right. The pupils were equal and reacted well to light.

On the second day after admission 0.5 gr. of papaverine was injected into the left common carotid artery.

Two days later another injection of 0.5 gr. of papaverine was made. During the next 10 minutes the patient moved his right arm spontaneously and on command

No change occurred in the right lower extremity. All movements of the right extremities, including withdrawal from painful stimuli, disappeared 10 minutes after the second injection. The following day, a single dose of 1 gr. of papaverine was injected into the left common carotid artery. No change in the neurologic status of the patient ensued. He died 24 hours later, following a slowly progressive worsening of his clinical status.

Necropsy revealed multiple infarctions of the left cerebral hemisphere. Thrombi could not be demonstrated in any of the major cerebral vessels or their branches.

Case 2. Mrs. B.J., a 67-year-old housewife, was admitted to Bellevue Hospital with a complete left hemiplegia which had developed gradually over the previous 72 hours. She had not lost consciousness and was mentally lucid. There was no aphasia. Neurologic study led to a diagnosis of cerebrovascular thrombosis and/or ischemic cerebral infarction. She failed to show any sign of improvement during the ensuing 2 weeks.

Therefore on the 15th day after admission, 0.5 gr. of papaverine was injected into the right common carotid artery. Within 1 minute the patient could voluntarily move the left lower extremity and could lift this extremity from the bed. No change was noted in the left upper extremity. During the next 30 minutes the left lower extremity became progressively weaker and at the end of that time voluntary motion of the left lower extremity was impossible. Four hours later a second right intracarotid injection of 0.5 gr. of papaverine was carried out. No neurologic change was noted. A third injection of 0.5 gr. of papaverine was made into the right common carotid artery the following day. No neurologic change was noted. This patient died 4 weeks following this study and permission for necropsy could not be obtained.

Case 3. F.C., a 56-year-old female, was admitted to our service with a complete right hemiplegia and a sensory-type aphasia. The hemiplegia had been gradual in onset, occurring over the course of 18 hours, and had been complete for 3 days prior to admission. Following complete neurologic evaluation the diagnosis of ischemic infarction of the left cerebral hemisphere was made and a trial of intracarotid papaverine was decided upon.

Therefore, 4 days following the onset of hemiplegia 0.5 gr. of papaverine was injected into the left common carotid artery. No objective neurologic change was noted during the next 30 minutes. A second 0.5 gr. of papaverine was injected into the left common carotid artery at the end of this 30-minute period and again no objective neurologic change could be noted. No further injections were made.

Case 4. B.J., a 65-year-old colored male, was admitted with a right hemiplegia of 3 hours' duration. The hemiplegia had occurred suddenly and was accompanied by giddiness and severe left-sided headache, but was not accompanied by loss of consciousness. Hemiplegia had been complete since the time of onset. Following complete neurologic and medical evaluation, the diagnosis of ischemic cerebral infarction in the left cerebral hemisphere, secondary to embolism from a cardiac mural thrombus, was made.

Forty-eight hours after onset of hemiplegia, 1 gr. of papaverine was injected in the left common carotid artery. Immediately upon completion of this injection painful stimuli applied to either the right arm or right leg resulted in withdrawal of these extremities. Withdrawal from painful stimuli had not been present previous to this injection nor at any time during the previous 2 hours that the patient had

been on our service. This withdrawal from painful stimuli persisted for the following 30 minutes but no further neurologic improvement was noted. Therefore, an additional 0.5 gr. of papaverine was injected into the left common carotid artery. No neurologic improvement was noted. No further injections were made. This patient was followed by us for the ensuing 2 weeks, during which he showed evidence of neurologic improvement including some return of voluntary motion to the right lower extremity and moderate improvement in his speech difficulty.

Case 5. R.Y., a 47-year-old white male, was first seen 48 hours after development of a right hemiplegia accompanied by motor-type aphasia. The hemiplegia had occurred gradually during the course of the previous 14 hours. Complete neurologic study led to a tentative diagnosis of ischemic cerebral infarction of the left cerebrum, secondary to cerebral vascular thrombosis.

On the second day after admission, 4 days after onset of hemiplegia, 1 gr. of papaverine was injected into the left common carotid artery. Upon completion of this injection, the patient withdrew the previously paralyzed extremities from painful stimuli and could weakly voluntarily move the right lower extremity. He could not voluntarily use the upper extremity. There was no change in reflexes. The voluntary motion of the right lower extremity lasted approximately 45 minutes. Withdrawal from painful stimuli of both extremities lasted approximately 2 hours. Further intracarotid injection was refused. The patient was followed for a period of 2 weeks and during this time no appreciable improvement was noted.

Case 6. A.R., a 67-year-old white female, was first seen 1 week following onset of left hemiplegia. There was no speech involvement. Neurologic evaluation led to a diagnosis of ischemic cerebral infarction probably on the basis of cerebral vascular thrombosis.

On the day after admission, 8 days following onset of hemiplegia, 1 gr. of papaverine was injected into the right common carotid artery. During a 2-hour period of observation following the injection no change in the neurologic status was noted. No further injections were made.

DISCUSSION

In summarizing our experiences, we found that in 4 of these 6 cases, immediately following intracarotid injection of papaverine, there was some objective neurologic change which could be interpreted as neurologic improvement. In 2 cases this was evidenced by voluntary motion of the previously paralyzed extremities. In the other 2 cases this was evidenced by withdrawal of the previously paralyzed extremities from painful stimuli. Subjective sensations were not recorded during this phase of the investigation and are not herein reported. In those cases in which objective improvement was noted, the improvement was transient in every instance.

During the early phase of our investigation one sobering experience occurred which deserves mention in a study such as this concerned with treatment of cerebral ischemic infarction. We were called to see a patient who had a hemiplegia of 24 hours' duration. The patient had been examined previously by the medical resident and the attending medical staff and by our neurosurgical resident, and each examiner had found the patient to be completely

hemiplegic on the right side and each had reached the diagnosis of cerebral vascular thrombosis. We examined this patient and found the same objective findings and reached the same clinical diagnosis. Therefore, the patient was prepared for intracarotid injection. Following sterile preparation of the cervical region for injection, the patient was asked to lift the right paralyzed extremity before the carotid puncture was carried out. This request was purely a theatrical one to demonstrate to observers of this procedure the paralyzed condition of this extremity. However, the patient promptly obliged by lifting the right extremity which previously had been completely paralyzed and which had demonstrated no voluntary motion or withdrawal from painful stimuli. This case is cited merely to demonstrate the dangers in evaluating results of therapy in this type of investigation and explains our reluctance to attempt to draw any conclusion from early findings in this study.

Several miscellaneous observations may be mentioned at this time. In 10 consecutive cases of cerebral arteriography in which diodrast was used as the contrast medium cerebral arteriograms were made before and after the intracarotid injection of 1 gr. of papaverine. In 6 of these cases there was objective evidence of dilatation of the medium-sized intracranial arteries in arteriograms after intracarotid papaverine as compared with control arteriograms. Uihlein[12] had previously noted similar effects of intracarotid papaverine on the caliber of intracranial vessels during arteriography. We have observed other phenomena that deserve further investigation. In 2 epileptic patients, the intracarotid injection of papaverine produced immediate onset of generalized convulsions. In 6 patients, the injection caused a sudden marked increase of peripheral systolic and diastolic blood pressure which lasted approximately 2 minutes. This is in marked contradistinction to the fall in blood pressure that usually accompanies intravenous administration of this drug. This rise in blood pressure cannot be ascribed to activation of the carotid sinus reflex since it was observed to occur in atropinized subjects. Whether it is secondary to dilatation of intracranial blood vessels, increased intracranial blood flow, increased intracranial pressure, direct effect of the drug upon vasomotor centers or other causes cannot be stated at this time. Similar changes in peripheral blood pressure have not been observed by us following intracarotid injection of 5 per cent glucose or of 35 per cent diodrast.

Many patients in whom intracarotid papaverine injection was carried out under local anesthesia complained of sudden severe burning of the ipsilateral side of the face and head, similar to, but apparently not as severe as, that occurring with intracarotid diodrast. One patient complained of burning in the face and contralateral fingers and became momentarily disoriented. In several instances marked flushing and cutaneous vasodilatation of the ipsilateral face followed intracarotid papaverine injection.

Obviously, conclusions are not warranted in a preliminary report of this type regarding therapeutic possibilities of intracarotid injection of

vasodilating substances. Certain factors can be pointed out, however, which indicate that further investigation in this regard may be fruitful. Despite the unquestioned action of papaverine as a vasodilating agent, its oral or parenteral use has not been attended by objective evidence of increased cerebral blood flow. On the other hand Henderson, Shipley and Chen[6] have shown that intra-arterial coronary artery injection of papaverine or its analogue paveril is attended not only by vasodilatation but also by marked increased blood flow in the dilated arteries. Mufson[9] has demonstrated the clinical efficacy and feasibility of intra-arterial administration of vasodilating agents in cases of peripheral obliterative arterial disease. The suggestive transient clinical improvement observed in 4 of our 6 cases, the enlarged caliber of cerebral arteries noted by arteriography following intracarotid papaverine, and the rationale of delivering such an agent directly into the vessels one wishes to dilate indicate that further exploration of this technique may prove fruitful. We are currently evaluating the use of a prolonged intracarotid drip of a solution containing papaverine as a means of producing a more prolonged vasodilating effect than single injections can provide.

SUMMARY

This constitutes a preliminary report of an investigation into the intracarotid injection of a known vasodilating agent. Objective neurologic improvement of a transient nature followed intracarotid injection of papaverine in 4 out of 6 patients with hemiplegia caused by ischemic cerebral infarction. The rationale of intracarotid administration of vasodilating agents has been outlined and possible avenues of further investigation in this regard have been indicated.

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CYTOLOGIC DIAGNOSIS OF TUMOR CELLS IN CEREBROSPINAL FLUID*

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THIS article deals with the study of 22 consecutive cases of cerebrospinal fluid examinations in which the referring physician requested that the fluid be examined for tumor cells. Tumor cells were identified in 5 cases. In 17 cases no tumor cells were found and in only 1 of these was the presence of an intracranial tumor (acoustic neuroma) later proved. In 2 others there are reasonable grounds for suspecting that a tumor may have been present but this, subsequently, has not been demonstrated. In the remaining 14 cases the existence of brain tumor has been ruled out. The technic used for the smear preparations was centrifugation, smearing of the sediment on previously albuminized slides, fixation, and staining by the Papanicolaou method.

The 5 cases in which tumor cells were identified in the cerebrospinal fluid are presented in the following reports.

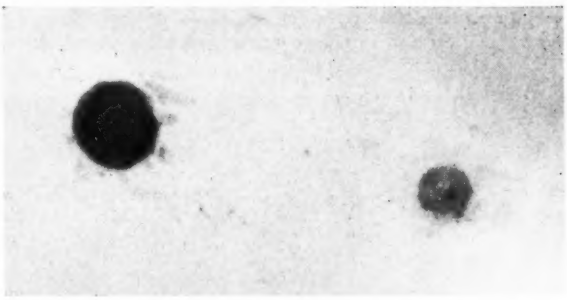

FIG. 1. Two large tumor cells, one partially degenerated.

CASE REPORTS

Case 1. C.S.D., a 61-year-old male, was admitted on Nov. 27, 1950 with fibrillary twitchings of the muscles of both hands and unilateral vocal cord paralysis. The CSF contained numerous tumor cells. Roentgenograms of the chest showed no abnormalities, and examination of the sputum for tumor cells gave negative findings. Ventriculography revealed an internal hydrocephalus with dilatation of the lateral and 3rd ventricles.

* Read at the Joint Meeting of the Northwest Region College of American Pathologists and the Pacific Northwest Society of Pathologists, Spokane, Washington, May 16, 1952.

Because of his critical condition no intracranial surgery was attempted and he expired Dec. 1, 1950.

Necropsy disclosed a small primary bronchogenic carcinoma in the upper lobe of the right lung with metastases to the brain and meninges.

Case 2. H.R., a 58-year-old male, was admitted March 19, 1951 with complaints of double vision and numbness of the right arm and right side of the face. Positive neurological findings were choked discs, paralysis of the left side of the soft palate, and impairment of pain recognition over the entire right half of the body. The CSF contained numerous large tumor cells suggestive of a metastatic origin (Fig. 1). Ventriculography disclosed a space-occupying mass in the brain stem above the 4th ventricle.

A polythene catheter was inserted between the right lateral ventricle and the cisterna magna to relieve the hydrocephalus. Temporary clinical improvement followed. A thorough search for the primary tumor was unsuccessful. Deep X-ray therapy to the head was ineffective and he expired on April 14, 1951.

Necropsy revealed a small highly malignant primary adenocarcinoma in the head of the pancreas with retroperitoneal and posterior mediastinal lymph node metastases. There was a solitary cerebral metastasis involving the dorsum of the pons and occluding the aqueduct of Sylvius.

Case 3. T.C., a 60-year-old man, was first admitted Dec. 28, 1951, when he presented signs and symptoms of a herniated lumbar nucleus pulposus which was excised with subsequent relief of symptoms. One month later he complained of severe headache, and bilateral choked discs were discovered. Roentgenograms of the chest showed mediastinal widening but no primary lung tumor. Cerebral angiograms revealed a left frontal tumor. Two CSF specimens were negative for tumor cells but a third specimen obtained after agitation of the lumbar subarachnoid fluid by repeated aspiration and injection contained numerous large bizarre tumor cells (Fig. 2).

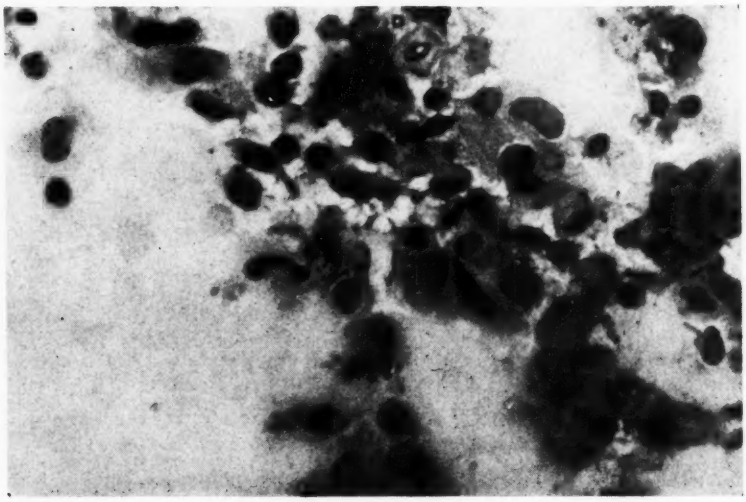

FIG. 2. Many bizarre tumor cells.

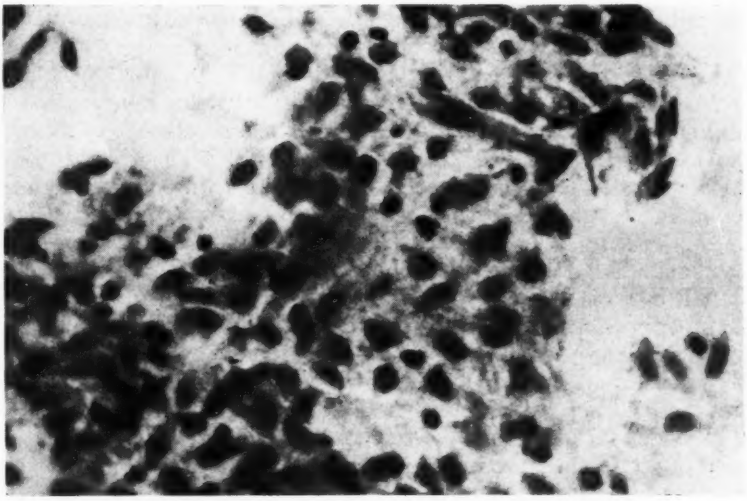

FIG. 3. Typical gliomatous pattern of a sheet of tumor cells.

Necropsy on Feb. 28, 1952 disclosed a tiny primary oat cell bronchogenic carcinoma in the upper lobe of the right lung with large mediastinal metastases. The brain contained numerous nodules of metastatic tumor, one of which had extended into the subarachnoid space.

Case 4. M.B., a 60-year-old woman, was admitted Jan. 22, 1952 with partial left hemiplegia and bilateral choked discs. Cerebral angiography revealed a mass in the right hemisphere. The CSF contained spindle-shaped cells suggestive of glioma.

A Grade III glioma (astroblastoma) was surgically excised.

Two months later she was apparently in good health, with no gross evidence of neurological defects.

Case 5. L.R., a 40-year-old male, was admitted Jan. 26, 1952 with a history of severe headaches for the past year. The only positive finding neurologically was a left homolateral hemianopsia. Cerebral angiography outlined a tumor in the right posterior parietal region. The CSF contained numerous tumor cells which were identified as being glial in origin (Fig. 3).

A Grade II glioma (astrocytoma) was surgically removed. After angiography and before the surgical procedure he suddenly stopped breathing and went into shock. Respiration resumed spontaneously after the skull was opened. Later complete anuria developed, and he expired on the 9th hospital day.

Autopsy findings included severe hemoglobinuric nephrosis, a recent Cushing's duodenal ulcer, and terminal pulmonary edema and pneumonia. There was some residual tumor about the edges of the operative site.

DISCUSSION

The propensity of certain malignant primary and metastatic tumors of the central nervous system to seed the meninges and ependyma has been well established. This type of case was first reported in 1907 by Spiller[4],

who described diffuse meningeal implants from an ependymoma of the 4th ventricle. Since this time there have been numerous reports of implants from various types of malignant gliomas as well as in cases of metastatic tumor, retinoblastoma, pinealoma, sarcomatosis of the meninges, and even of meningiomas with sarcomatous transition.[1] The percentage of malignant gliomas with meningeal involvement varies from 47.6 per cent in the medulloblastoma group to 7.1 per cent in the cases of more adult astrocytomas.[3] These implants may often be present in the meninges of the spinal cord, as observed by Svien, Gates, and Kernohan[5] in 31.5 per cent of their cases of ependymoma. The seeding is probably a direct result of exfoliation of the tumor cells into the cerebrospinal fluid with transplantation of these viable cells to other portions of the meninges. Any tumor capable of exfoliation can lead to the finding of tumor cells in the spinal fluid if either the ependyma of the ventricles or the leptomeninges are involved. The action of gravity explains the presence of these exfoliated cells in the lumbar spinal fluid and, in the absence of a block in the cerebrospinal fluid circulation, they should be expected to be most numerous in this dependent fluid. Agitation of the lumbar subarachnoid fluid by repeated aspiration and reinjection may yield tumor cells in cases in which aspiration alone has been unsuccessful. Fluid aspirated from cerebral cysts may also contain tumor cells.[2] In some cases it may be possible to determine from the appearance of the tumor cells whether they arise from a primary or metastatic neoplasm. Finally, it has been reported by Willis[6] that disseminated carcinomatosis of the spinal meninges can occur without any grossly demonstrable metastatic neoplasm in the central nervous system. This may result from minute, undiscovered, blood-borne metastases in the ependyma or meninges, or from meningeal infiltration by way of nerve sheath, nerve root, or perineural lymphatic extension.

SUMMARY

1. The results of spinal fluid examination for the presence of tumor cells in 22 consecutive requests for this procedure have been presented.

2. In 5 instances the fluids were reported as containing tumor cells: 3 from metastatic carcinoma, and 2 from primary glioma.

3. In 1 instance the tumor cytology was considered diagnostic of metastatic carcinoma rather than a primary central nervous system tumor.

4. In 1 patient two spinal fluids were negative for tumor cells but a third specimen obtained after agitation of the lumbar subarachnoid fluid contained numerous bizarre tumor cells.

5. In the remaining 17 spinal fluids which were reported as negative for tumor cells there was 1 case of proved acoustic neuroma, which would not be expected to exfoliate, and 2 cases in which tumor was strongly suspected but not definitely proved.

6. In our opinion spinal fluid examination is a useful adjunct in the differential diagnosis of intracranial neoplasm from other lesions of the central nervous system.

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EXPERIMENTAL CEREBRAL CONCUSSION*

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SINCE the incidence of closed head injury seems to be assuming increasing proportions in modern high-speed life, a better understanding of the phenomenon of cerebral concussion is needed. Unfortunately, relatively little basic information is yet available regarding the detailed pathophysiology of cerebral concussion.[2]

The mechanics of brain trauma have been investigated rather extensively and Denny-Brown and Russell[4] have pointed out that both compression and acceleration concussion can occur although the latter is responsible for the majority of cases of concussion seen in man.

The appearance of large amounts of free acetylcholine in the spinal fluid of experimental animals following concussion has been described by Bornstein.[1] He pointed out that while free acetylcholine is never normally present in cerebrospinal fluid, it is found in relatively large quantities shortly after head trauma. He also felt there was a positive correlation between the concentration of acetylcholine in the cerebrospinal fluid, the clinical signs of concussion and the postconcussion EEG changes. Tower and McEachern[10] have studied these factors following closed head injury in the human and demonstrated low cholinesterase activity of the CSF as well as free acetylcholine which might be present in large amounts. Recovery was associated with reversal of these changes. These findings suggest a rationale of therapy specifically directed at the reversal of one of the sequelae of trauma of the brain. In experimental animals, Bornstein[1] found that both the EEG pattern and the stuporous condition could be abolished by appropriate doses of atropine sulfate (0.5–1.0 mg./kg.). The extension of this anticholinergic therapy to man has been carried out by Ward,[12] who reported the use of large daily injections of atropine sulfate in doses of 0.1 mg./kg. (gr. 1/10 in adult man) in an initial series of patients who had sustained very severe closed head injuries. He described dramatic and consistently reproducible clinical improvement in selected instances.

If this formulation of the role of acetylcholine in closed head injury is valid, experimental verification of two sets of data should be possible. The first of these is the manner in which acetylcholine is released follow-

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This study was supported (in part) by funds provided under contract AF 18(600)67 with the USAF School of Aviation Medicine, Randolph Field, Texas.

ing cerebral concussion. Walker, Kollros and Case[11] have presented experimental evidence that would indicate that the clinical manifestations of experimental concussion are the result of intense neuronal excitation of the central nervous system at the moment of the blow to the head. Bornstein[1] has postulated that the appearance of the acetylcholine may be the result of this intense neuronal excitation. To gain further insight, detailed studies of cortical and subcortical electrical activity during and following concussion were therefore carried out.

The second set of data deals with the effects of circulating acetylcholine so produced, and specifically with its possible relation to so-called cerebral swelling. White *et al.*[14] reported an increase in volume of the concussed cat brain associated with a moderate rise in cisternal pressure within 4 hours of concussion. Acetylcholine is a powerful vasodilator and may alter vascular permeability. It is conceivable that its presence in high concentration may be a factor in this increase in brain volume. Since a marked change in brain volume should cause a change in spinal fluid pressure, continuous recordings of spinal fluid pressures were made during and following concussion.

As a part of a broad investigation of cerebral concussion, this preliminary report deals with these two factors.

MATERIAL AND METHODS

Acceleration concussion was produced in 20 animals (19 cats and 1 *Macacus rhesus* monkey). The electrical activity of the cortex and subcortical structures, respirations, EKG, blood pressures, and spinal fluid pressures were recorded during and following acceleration concussion.

Production of Concussion. A gas pressure gun was designed and constructed to produce acceleration concussion (Fig. 1). It has a main chamber loaded with compressed nitrogen gas up to 100 lbs./square in. A piston-like plunger is contained in a barrel leading off from this chamber, and is held in position by a trigger mechanism. On release, piston velocities up to 45 ft./sec. are obtained. Acceleration up to 2500 ft./sec. is possible with interchangeable piston units. A built-in compression head on the barrel stops the piston within 3½ cm. after contact with the animal's head. This achieves an almost instantaneous blow, the minimum impact duration being 9 msec. with a piston velocity of 18 ft./sec. In all experiments, the weight of the piston was greater than the head of the animal so that factors of inertia may be disregarded. Cats of identical body weight (3 kg.) were used with a piston velocity of 18 ft./sec. The blow

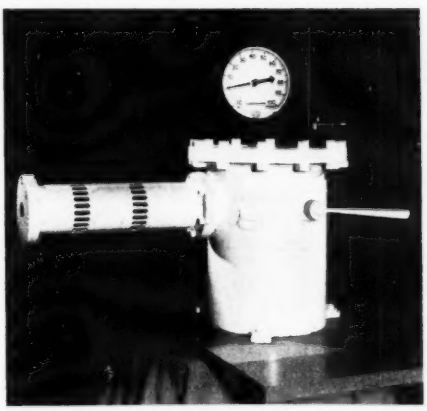

FIG. 1. Concussion gun.

was delivered to the parietal area of the intact skull with the head freely suspended and the body supported in a metal stand which could slide freely on a metal surface. When anesthesia was used, either Vinethene or Dial* was employed, and in all cases a tracheotomy tube was used to insure an adequate airway.

Electrographic Recording from Cortical and Subcortical Structures. The electrical activity of the cerebral cortex was recorded by means of small dural electrodes fixed in fine drill holes through the calvarium, or by means of tiny skull screws (Fig. 2). The shielded leads from these electrodes were carried subcutaneously in a single cable to the nape of the neck, where a plug connection was made to a second cable passing to an 8-channel Grass electroencephalograph. The 6 cortical electrodes were placed in a constant position 2 cm. apart in all animals. In most cases short time constants were used in recording for technical reasons. The electrical activity of subcortical structures was obtained by means of electrodes placed in various portions of the brain stem by means of a stereotaxic instrument. These electrodes consisted of either No. 30 nichrome steel wire or a fine No. 36 enameled copper wire electrode implanted stereotaxically in the same regions by means of a No. 22 needle which is then withdrawn, leaving the silver-tipped copper wire in place. These electrodes were fixed by dental cement as they ran through a small drill hole in the skull. The position of the recording tip was verified in each case by means of sections of the formalin-perfused brain. The electrocardiogram was recorded on the electroencephalograph from needle electrodes in an upper and lower extremity. The respirations were recorded on the electroencephalograph from a thermocouple fixed in the tracheotomy tube.

FIG. 2. Skull (cortical) and subcortical electrodes before scalp closure.

Pressure Recordings. Both blood and spinal fluid pressures were recorded by means of Statham strain gages, the output of which was amplified by a carrier amplifier and ink recording. Blood pressure was recorded through a P.E. 50 polyethylene catheter threaded up the carotid artery. Both the catheter and the chamber in the Statham strain gage were filled with a 1 per cent solution of Paritol C† in saline to prevent coagulation of blood. Spinal fluid pressures were recorded through a No. 10 nylon catheter tied into the lumbar subarachnoid space at the time of laminectomy and threaded up the spinal canal to the mid-dorsal level. The recording system was filled with saline.

RESULTS

The criteria used to determine the presence of concussion in these experiments were essentially the same as those proposed by Denny-Brown and

* The authors wish to thank Ciba Pharmaceutical Products who kindly furnished this drug.
† The authors wish to thank Wyeth Inc. who kindly furnished this drug.

Russell.[4] These signs of concussion were identical whether anesthesia was used or not, and consisted of an abolition of the corneal reflex, loss of pinna reflex, abolition of respiratory regularity, cessation of spontaneous swallowing and licking movements, and loss of somatic musculature movements and pupillary reactivity. The corneal and pinna reflexes were found to be the most reliable indices and were tested every second following the blow until they had returned to normal. In certain animals, more than one concussive blow was administered so that data are available on 24 concussions in animals. With a piston velocity of 18 ft./sec., which was used in the majority of experiments, clinical concussion so defined lasted from 2 to 15 min. All cases in which anatomical damage could be demonstrated at autopsy, including subarachnoid hemorrhage, skull fracture, cortical contusion, and intracerebral petechial hemorrhages, were excluded from this series. Two deaths occurred in which the cause of death could not be determined on the basis of anatomical changes.

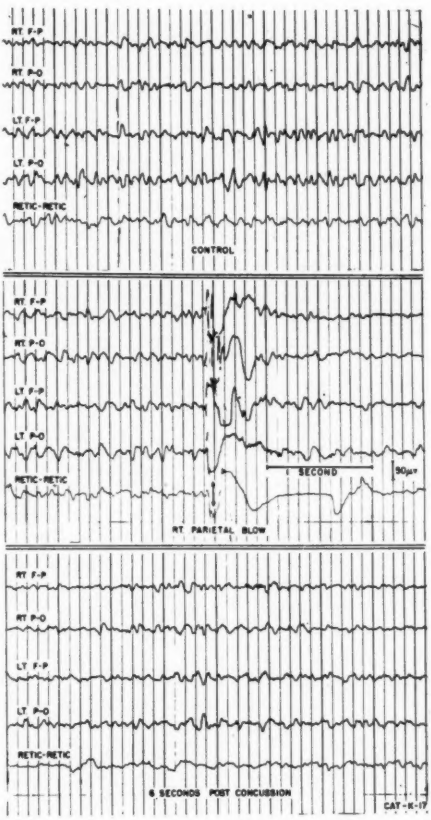

FIG. 3. High speed electrographic recording during concussion.

Electrical Activity. The electrical activity of the cerebral cortex was continuously recorded during and immediately following the blow in all instances, as well as intermittently up to a maximum of 12 hrs. following concussion. The technical problems were rather formidable since the recording leads were also subjected to the acceleration forces applied to the head. If no precautions were taken, amplifier blocking for periods of 5 to 10 sec. prevented reliable recording during this critical period of time. However, adequate shielding of leads which were collected into a single shielded cable which ran subcutaneously to the base of the cervical spine made it possible to obtain useful recording within a fraction of a second of the moment of impact.

Changes in the electrical activity of the cortex during the first 10 sec. following the blow were uniform in all cases. Coincident with release of the piston there is an immediate spike artifact present in all channels which ap-

pears to be secondary to the shock wave or noise from the compressed gas gun. In no instance was any increase in cortical activity seen during the first 10 sec. following the blow. This is best seen in Fig. 3 where adequate recording was obtained within a fraction of a second of the blow. Nothing resembling seizure discharges was seen at any time regardless of the presence or absence of anesthesia.

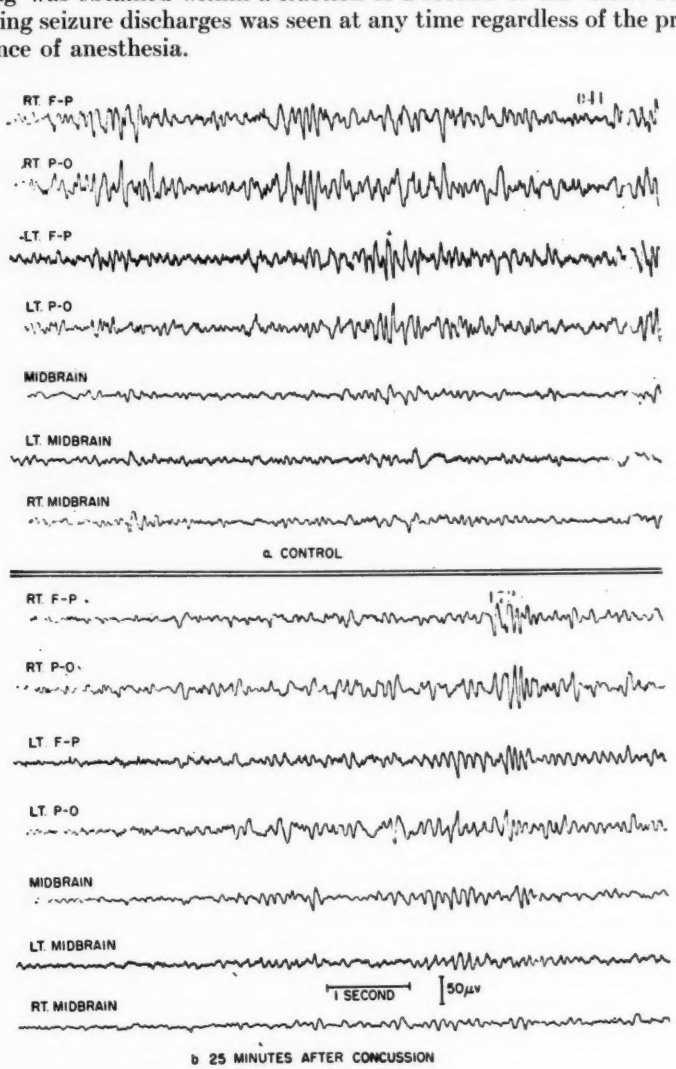

FIG. 4. Cortical and subcortical recording before and after concussion (cat, light Vinethene anesthesia).

Changes in cortical electrical activity were, in general, surprisingly minimal in view of the massive change in physiological function. In certain cases, no definite changes in cortical activity could be observed and in 1 instance the cortical activity was unchanged immediately following concussion although the corneal reflexes were absent for 90 sec. However, in the majority of instances a generalized flattening appeared immediately after the blow which consisted primarily of a moderate to marked reduction in amplitude, often with no major change in the frequency. This flattening was occasionally greater on the side of the impact and persisted for 10 min. up to several hours. Return of cortical activity to control levels occurred gradually as a rule, though in some cases the recovery was dramatic and abrupt. In Fig. 4b, marked flattening is still present on the left of the record 25 min. after concussion. This is abruptly replaced by normal activity in the center of the record. In Fig. 5, similar cortical flattening is present bilaterally. This appeared immediately following a 4-sec. postconcussive episode of slow wave activity and demonstrates selective voltage reduction on the faster frequencies. In animals anesthetized with Dial, the Dial bursts were abolished by concussion and reappeared at times varying from 10 sec. to several minutes after the blow. The cortical Dial bursts seemed to be more resistant to concussion effects than any other feature of the cortical record. Abnormal slow waves, such as those described in man, though occasionally

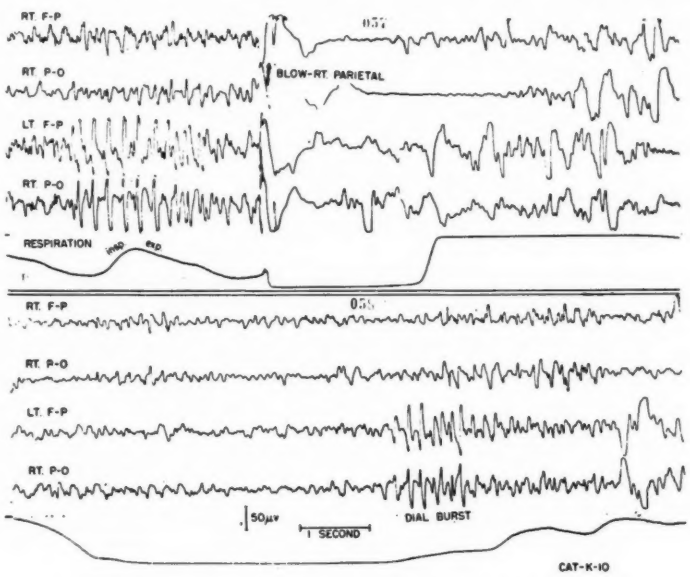

FIG. 5. Cortical and respiration recording during and following concussion (cat, light Dial anesthesia).

seen (Fig. 5), were not a prominent feature of the records obtained in animals with threshold concussion.

Recording of the electrical activity of subcortical structures was carried out in 10 instances. Structures whose activity was recorded during and following concussion included the medial and lateral reticular formation just below the red nuclei of the midbrain (Figs. 3 and 4), periaqueductal grey, basis pedunculi and substantia nigra. The electrical activity paralleled that seen in the cortex. The one dramatic exception to this was the activity recorded from the medial reticular formation, which always showed far greater and longer depression of electrical activity following concussion than cortical and other subcortical structures studied. Occasionally other structures in the

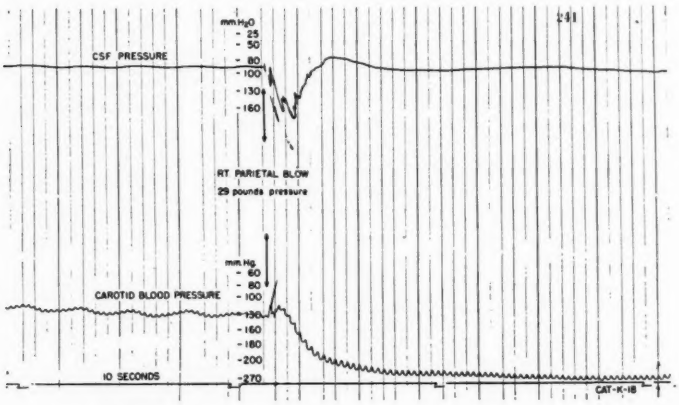

FIG. 6. Spinal fluid and carotid blood pressure records during concussion.

reticular formation showed an actual increase in electrical activity during recovery (Fig. 4), per second activity occurring in short bursts. This was not seen in records of recovery in the cortex.

Spinal Fluid Pressures. The pressure of the cerebrospinal fluid is inevitably difficult to reliably record during impact because of the physical effects of momentum on any hydraulic recording system. This instrumental artifact merges with a recorded 2–3 sec. episode of sharp pressure changes incident to joggling of the head following the blow during fixation of the head on the head rest. Two seconds following the blow, a transient, moderate rise in spinal fluid pressure occurred (Fig. 6). This rise never exceeded 80 mm. of water and never lasted more than 2 sec. During the next 2 sec., the peak of the pressure then fell rapidly to a point slightly below the preconcussion control pressure, but returned rather quickly to normal. Continuous recording of the spinal fluid pressure for as long as 5 hrs. subsequent to concussion in 12 cats failed to show more than a 10 mm. H_2O change in this pressure. Simultaneous recording of carotid blood pressure (Fig. 6) demonstrated a rather precipitous rise in blood pressure occurring during the first

2 sec. after concussion. This hypertension persisted for 15 to 25 sec. at which time it gradually returned to preconcussion levels. It should be noted that the blood pressure may increase over 100 mm. Hg while the spinal fluid pressure is actually falling (Fig. 6). The vagus nerves were not sectioned in any of these experiments and consequently a postconcussive bradycardia of varying degree was always seen. In spite of the cardiac slowing, the postconcussive rise in blood pressure was still a remarkably constant phenomenon.

Respiratory changes at the time of concussion consisted of temporary inspiratory or expiratory apnea as recorded by a thermocouple implanted in the tracheotomy tube (Fig. 5).

DISCUSSION

The critical mechanical factors involved in concussion have been studied in detail by Denny-Brown and Russell[4] who pointed out that acceleration (either positive or negative) is the adequate stimulus for the production of concussion. It is thus necessary to clearly distinguish between acceleration concussion in which the skull is subjected to a sudden change in velocity, and compression concussion in which the skull is compressed and distorted enough to cause a rise in intracranial pressure to levels greater than 300 mm. of mercury. Experimental variations of compression concussion can be devised in which the intracranial contents are directly compressed through an opening in the skull in a variety of ways. However, it is acceleration concussion that commonly occurs in the human and this differs fundamentally from the various types of compression concussion. Miller[7] postulated that concussion produced transitory compression anemia, but the data of Denny-Brown and Russell[4] on venous outflow and intracranial pressure changes do not substantiate this hypothesis. For that reason, acceleration concussion was used in these experiments, even though it may not be technically desirable to have both animal and recording system moving at a velocity of 18 ft./sec. for short periods of time.

The changes in electrical activity of cortex and brain stem structures immediately following the moment of impact fail to confirm the findings of Walker, Kollros and Case.[11] These authors reported that there is an initial electrical discharge at the moment of impact ranging in voltage from a few hundred microvolts to a millivolt or more. Following this initial discharge, they also described a marked increase in the frequency of cortical activity with little decrease in amplitude. This spiky, fast cortical activity was likened to that seen during a major convulsive seizure. In this series, such discharges were not seen, even in the non-narcotized animal. However, acceleration concussion was used in the present experiments while Walker *et al.* produced concussion by means of a blow delivered by a weight falling through a glass tube upon a column of water in contact with the intact dura mater, i.e. compression concussion. It could well be that this fundamental difference in the phenomena studied would account for the differences noted.

The changes in electrical activity of cortex here reported largely agree with those described by Williams and Denny-Brown,[15] who also were studying acceleration concussion. However, blocking of amplifiers prevented them from obtaining records during the first second following the blow and thus they presented no data regarding possible seizure discharges during this early period.

Changes in the electrical activity of subcortical structures (hypothalamus, red nucleus and cerebellum) during acceleration concussion have been studied by Spiegel *et al.*[9] They reported that the disturbances in these structures are similar to those seen in cortical records and in some instances the subcortical changes may be even more intense than those seen in the cortex. Recording from different portions of the brain stem in the present experiments would largely agree with their findings. The secondary electrical activation seen in the lateral reticular formation in our experiments during the recovery phase is largely unexplained. It is of great interest, however, that the electrical activity of the medial reticular formation in the midbrain always showed greater depression of electrical activity than did the cortex (Fig. 3). The reduction in amplitude in other brain stem structures largely paralleled those seen in the cortex. This observation correlates well with the findings of Rhines, Magoun, and Windle,[8] who found that the patellar hyperreflexia seen in cats after concussion is temporally associated with a rise in threshold to stimulation in the medial reticular formation.

One might postulate that these findings play a role in explaining the marked clinical changes associated with concussion since these clinical changes may occur in the absence of any definite change in cortical activity. We know that these portions of the reticular formation play a role in the maintenance of the waking state and consciousness. Thus selective depression of these structures might be playing a role in the coma that is associated with concussion. It is not clear why these circuits should be selectively depressed by these critical acceleration forces. Although no evidence is available, one might postulate that the collaterals to this region from the ascending sensory system, which maintain activity in these ascending activating systems, are more vulnerable because of their small diameter and lack of myelination. Further specific studies on this point are needed. However, it is evident from all available data that changes in purely cortical function are not the basis for the loss of consciousness, reflex depression and other signs of clinical concussion as seen in the human. The maximal physiological changes in such patients must be postulated to occur in as yet incompletely delimited subcortical structures.

White, Brooks, Goldthwait and Adams[14] have carefully demonstrated that acceleration concussion produces an increase in brain volume up to a maximum of 5.5 per cent and that the degree of brain swelling varies directly with the severity of concussion. It appears in about 15 min., reaches a maximum in 5 to 24 hrs., and subsides after 3 days. They postulate that this is caused by an extravasation of fluid through the capillary walls. They also

noted that there is a slight to moderate rise (60 mm. of water) in cisternal pressure after concussion and during the period in which the brain is swollen, though these pressures were recorded only intermittently. A rise in intracranial pressure was not found to accompany acceleration concussion as reported by Denny-Brown and Russell,[3] which is in agreement with Webster and Freeman,[13] whose observations indicate that the cerebrospinal fluid pressure was not significantly elevated in most instances of cranial injury, either in the experimental animal or the human. They reported a direct correlation between the quantity of blood in the spinal fluid and the increase in spinal fluid pressure that occurred. The present experiments failed to demonstrate any long lasting change in spinal fluid pressure greater than 10 mm. of water during the first 5 hrs. following acceleration concussion. Elevations in spinal fluid pressure were seen in this experimental series, but only in those animals who had sustained anatomical damage. On the basis of this and previous data, one might assume that the presence of increased spinal fluid pressure indicates that more than pure concussion has occurred and that actual anatomical damage has been produced. In the present work, there was no evidence to suggest the presence of acute cerebral edema. That such edema can occur has never been demonstrated by quantitative chemical techniques, and recent studies by Eichelberger, Kollros, and Walker [5] have shown that the total nitrogen, water, and electrolyte concentrations in the concussed dog brain show no changes indicative of cerebral edema, either in the extracellular or intracellular compartments. The possible interrelationship of cerebral swelling or cerebral edema and intracranial pressure have unfortunately never been quantitatively studied.

The data obtained unfortunately throw no direct light on the mechanism of liberation of acetylcholine. It would seem that, in the absence of confirmatory evidence, one cannot postulate that a massive seizure-like discharge is the responsible factor. Moreover, since Lorente de Nó[6] has shown that acetylcholine is released from peripheral ganglia by mechanical damage alone, it would seem that similar mechanisms might be operating during concussion.

Since acetylcholine is a vasodilator, the hypothesis could also be advanced that a vasodilation should occur following concussion and that such vascular changes might be reflected by changes in the cerebrospinal fluid pressure. No changes in pressure could be shown in these experiments.

It is obvious that further discrete data are needed regarding the role that acetylcholine may play in the genesis of the sequelae of closed head injury. The present therapy with atropine in human cases is also handicapped by the fact that only minimal doses are possible because of the peripheral, muscarinic actions of this drug. Whereas the effective dose of atropine sulfate in the animal is 0.5–1.5 mg./kg., the maximal dose used thus far in humans is approximately 0.1 mg./kg. Obviously an anticholinergic drug is needed that will be maximally effective centrally and will have minimal or no peripheral action. It is hoped that future experiments will permit development of such a method of therapy.

CONCLUSIONS

1. Acceleration concussion was studied in cats and monkeys with continuous recording of the electrical activity of cortex and certain brain stem structures, spinal fluid and blood pressures, and EKG and respirations.

2. The electrical activity of the cortex was reduced in amplitude following concussion with little change in frequency and at times essentially no change was produced.

3. At no time was any massive epileptic-like discharge observed during the first few seconds after the blow. Such a process thus can not be the mechanism of release of the acetylcholine that appears in the spinal fluid following concussion.

4. Changes in the electrical activity of brain stem structures largely paralleled those in the cortex except for the activity of the medial reticular formation where consistently greater depression of amplitude was seen as compared to cortex. The possible role of this phenomenon in the genesis of coma is discussed.

5. No change in spinal fluid pressure greater than 10 mm. of water was recorded during the first 5 hours following pure concussion.

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INTRA-ARTERIAL PRESSURES IN THE NECK AND BRAIN

LATE CHANGES AFTER CAROTID CLOSURE, ACUTE MEASUREMENTS AFTER VERTEBRAL CLOSURE*

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IN THE past six years we have carried out measurements of intra-arterial pressure in the carotid vessels in the neck and more recently in the intracranial arteries as well. Brackett and Mount[2] and Woodhall, Odom, Bloor and Golden[8] have reported similar studies. The intracranial studies have been facilitated greatly by the addition to our Sanborn electromanometer of a system to provide continuous minimal flushing with normal saline of the minute No. 27 gauge needle we use.† Pressure readings take place during this flushing process without introduction of inaccuracy and the problem of clotting is virtually eliminated even though we now dispense completely with anticoagulants such as heparin.

We have already reported[1] on a comparison between the pressures in the cervical carotid and those in intracranial arteries down to 0.4 mm. in diameter. The pressures measured in the neck and in the main intracranial branches of the internal carotid with the flow free reveal that there is virtually no measurable pressure drop in the anterior and middle cerebral trunks. Even in the small arteries on the superolateral surface of the hemisphere the systolic pressures run from 65 to 92 per cent of those in the cervical carotid with an average of 83 per cent in the twelve vessels measured in as many patients. Upon occlusion of the common carotid the percentage drop in pressure in all portions of the internal carotid and its accessible branches is the same. These measurements include some in arteries as small as 0.4 mm. in diameter. Hence measurement of the pressure drop in the carotid in the neck proves to be a reliable indicator of the degree of fall throughout the branches of that carotid's tree, and cervical carotid ligation has essentially the same effectiveness in the treatment of an intracranial aneurysm whether it arises from the carotid trunk or one of its smaller branches. Furthermore, since cervical carotid occlusion usually lowers the arterial pressure distal thereto by about

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† The Sanborn electromanometer with equipment for continuous flushing is obtainable from the Sanborn Instrument Co., 39 Osborne St., Cambridge, Mass.

50 per cent, this tactic appears to represent the most rapidly controllable means of achieving hypotension in the distribution of the internal carotid artery and may prove to be the most satisfactory means of producing hypotension to facilitate surgical removal of haemorrhagic lesions in this part of the brain.

We wish to present now: (1) serial measurements of cervical carotid pressure in the neck from the time of occlusion up to several months thereafter and (2) findings on the distal pressures in the vertebral artery and its branches before and after occlusion of this main vessel.

CAROTID PRESSURES LONG AFTER OCCLUSION

It has long been established that collateral circulation to the brain of man develops promptly after one carotid is partially occluded. Thus Dandy[4] found that his patients tolerated complete closure of the carotid in the neck within a week of a first operation at which such closure evoked neurologic signs of cerebral ischemia. Braden[3] has recently reported 3 patients in whom intracarotid pressures rostral to closure of the common carotid had returned to the original level when measured 2 years, 48 hours and 24 hours respectively after the closure. In each patient there had been a substantial drop acutely immediately after occlusion.

It would appear valuable in ascertaining the efficacy of carotid ligation to know how long the falls in pressure may be expected to persist. We have data bearing on this point in 9 patients with intracranial aneurysms.* In 8 of them we have a set of pressure measurements at the time of the original exposure of the vessels in the neck, and another set 6 days to 24 weeks later when the wound was reopened for additional occlusive measures. In a ninth patient we have only the late reading nearly 2 years after total occlusion.

In the following 2 cases complete closure of a carotid artery was not carried out at the first operation.

Case 1. In Donalda B., aet. 40 years, the interval between measurements was 6 days. At the first session complete common carotid occlusion had reduced the pressure from 130/95 to 30/28 and additional occlusion of external carotid had no further effect. A residual systolic pressure of only 23 per cent of that with the flow free may be followed by a late thrombosis spreading from the aneurysm into the main arterial branches (see our case G.D.M. in Sweet, Sarnoff and Bakay[7]). Hence we should not have employed complete occlusion here even if the patient had tolerated it for 30 minutes. However, in this patient, we had to open the occluding tantalum band almost completely in order to avoid the appearance of neurological signs. The pressure distal to it at that time was the same as with the flow free. Six days later the intracarotid pressure was about the same, 132/110, but now common carotid occlusion caused less reduction, to 63/58, or a remaining systolic level 48 per cent of the original. The addition of external carotid occlusion produced no further change and the patient remains well after permanent closure of the common carotid.

* For the opportunity of obtaining some of these readings, we are greatly indebted to Dr. John Drew, Chief of the Neurosurgical Service, Veterans Administration Hospital, Boston, Mass. and to Dr. Hannibal Hamlin, Clinical Associate in Surgery, Massachusetts General Hospital, Boston, Mass.

Case 2. In Ann B., aet. 54 years, the initial readings were: with flow free 142/85, with common carotid closed 59/38, and with common plus external closed 59/38. Although the residual systolic pressure was 42 per cent of the original, signs of cerebral ischemia were apparent within 10 minutes of clamping the common carotid. This patient was left with the artery about half closed by the tantalum band, a degree of closure that had no effect on the systolic pressure. At the reopening of the neck, 11 days later, there was extreme fluctuation in the blood pressure distal to total occlusion of the common carotid, which we shall describe below. Complete occlusion of both common and external carotids has been well tolerated.

In the next 7 patients the common carotid artery was completely occluded at the first procedure.

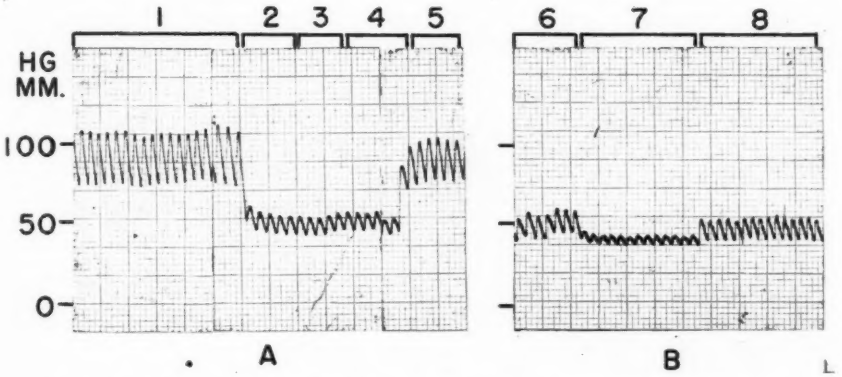

FIG. 1. *Case 6.* Pressure recording in right common carotid artery. (A) 1, 5=free flow, 2, 4=proximal occlusion of common carotid artery, 3=additional occlusion of external carotid. (B) Recording in distal part of the same artery 3½ months after proximal occlusion. 6, 8=no significant change in pressure drop as compared with 2, 3, 4. 7=effect of manual compression of contralateral carotid artery.

Case 3. In Margaret S., aet. 40 years, the period intervening between the two operations was 12 days. The percentage drop distal to occlusion remained the same although the systemic systolic pressure was 175 mm. Hg the first time and only 140 at the next session.

Case 4. In Floyd G., a 28-year-old man, whose two measurements were made 5 weeks apart, essentially the same maintenance of percentage drop ensued.

Case 5. In Samuel C., a 32-year-old male, a major fall to 33 per cent of the initial level followed common carotid occlusion acutely. Six weeks later this level had risen somewhat, but only ¼ the way to the general systemic arterial pressure. The actual figures were a fall from 118 to 39 mm. Hg acutely with a recovery to 62 mm. Hg in 6 weeks, so that a substantial reduction was still present. In this patient the rise in pressure was the result of the development of intracranial collateral channels because the level remained at 62 mm. Hg. when the external carotid was closed.

Case 6. In Agnes S., a 31-year old woman, the identical degree of pressure drop was maintained for the 3½ months that elapsed between the two studies (Fig. 1).

Case 7. In Eleanor W. (Fig. 2), a 65-year-old hypertensive arteriosclerotic female, signs of cerebral ischemia developed when her common carotid artery was occluded completely, as one might expect from a pressure drop of 216 to 54 mm. External carotid occlusion caused no further change. The common carotid was then partially closed by a tantalum band, but in the succeeding 9 weeks scar tissue completed the closure of this artery. Measurement then showed that the maximal systolic pressure distally had risen to 77 mm. This was shown to be caused by collateral back flow from the external carotid artery since the pressure fell almost exactly to 54 again when the latter vessel was occluded.

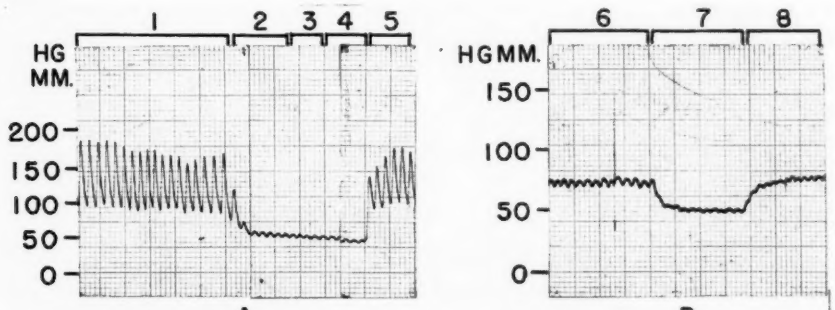

FIG. 2. *Case 7.* Pressure recording in right common carotid artery. (A) 1, 5 = free flow; 2, 4 = proximal occlusion of the artery, 3 = additional occlusion of external carotid artery. (B) Pressure distal to occlusion 10 weeks later shows some increase (6, 8) since previous recording; occlusion of external carotid artery reduces pressure to original level (7).

In contradistinction to the maintenance of a marked reduction in pressure in the previous 5 cases, is the behavior of our next patient.

Case 8. Robert K., aet. 35 years, showed pressure of 144/89 with the flow free, 69/58 with the common carotid occluded and 63/54 with common plus external occlusion. Five and a half months later his systemic arterial pressure was essentially the same but the pressure above complete occlusion of the internal carotid had now risen strikingly to 127/95. The residual pressure had come up from 44 per cent to 84 per cent of the systemic arterial level.

Case 9. In Elvira P., another 60-year-old hypertensive woman, the initial occlusion of the common carotid artery was carried out elsewhere without pressure measurements. Twenty-two months later, following a second subarachnoid haemorrhage, the carotid in the neck was exposed again. The systolic pressure distal to the ligation was 71 per cent of the systemic pressure—125 and 175 mm. Hg respectively.

Comment. In 10 of the first 38 patients in whom we made such pressure measurements acutely we found the levels distal to the occlusion of the internal carotid to be greater than in 60 per cent of those with the flow free. Consequently there is about a 25 per cent chance that the figure of 71 per cent in our Case 9 is nearly what it was immediately after the first operation.

In summary one notes that while the majority of this small series showed only slight to moderate late rises in the carotid pressure distal to full occlusion, the patients in our Cases 2 and 8 had a pronounced recovery of their pressures amazingly close to the previous level. The first two patients (Cases 1 and 2) who did not tolerate complete occlusion initially, but who did so 6–11 days later when their pressures distal to occlusion were higher, demonstrate another point, namely that the prompt development of an improved collateral circulation does not require a high degree of occlusion of a trunk artery.

SERIAL MEASUREMENTS DURING OPERATION

In view of the fluctuations seen in our late measurements we have made many more records during the initial 30–60 minute period of trial occlusion. In our first paper on this subject (Sweet and Bennett[6]), we found in a few instances that the pressure drops on occlusion were maintained at the same level 30 minutes later. The cumbersome apparatus we used at first made serial measurement difficult, but with the electromanometer we have made such studies, recently, in 8 patients. In 4 of these the pressures were almost exactly the same at the end as at the beginning of the period of common or common plus external carotid occlusion. In our Case 1, we studied the effect of bilateral cervical sympathetic block with xylocaine, since occlusion of common carotid alone or common plus external had produced such a marked drop in pressure to 30/28. The effective block appeared to improve slightly the collateral supply via the external carotid since the pressure with common alone occluded now rose to 40/35, while it remained at the original level distal to occlusion of the internal carotid. However, a conclusion that the sympathetic block improved the circulation rests on dubious grounds because there was a similar or even greater degree of spontaneous rise in 3 other patients.

The most striking of these spontaneous changes occurred in Case 2 during the patient's second operation. At the start of this the distal pressure fell from 117/50 to 50/30 when the common carotid was occluded, a systolic drop to 43 per cent of the original. But after 30 minutes of such occlusion the distal pressure had risen to 83/50. During the next 20 minutes remarkable fluctuations occurred; at one point a level of 118/65 was reached, and at the end of this period of observation the distal pressure was 90/55. The systolic and diastolic pressures as recorded from a brachial cuff were relatively constant during this time and there was no obvious change in the patient's general status. She was fully rational, cooperative and free of pain throughout. The addition of external occlusion on several occasions did not change the immediate reading obtained. In patient Beatrice S., a pressure of 150/97 dropped to 44/42 upon occlusion either of common and external carotid or of the former vessel alone. After 30 minutes the pressure had risen to 72/58; some of this rise was caused by retrograde flow via the external carotid as shown by a fall to 63/53 when that vessel was closed. This rise was not main-

tained and at 45 minutes the pressure distal to occlusion of the common carotid only had dropped to 56/45. Blood pressures at a brachial cuff recorded nearly constant throughout this time. Patient Helen N's closure of the common carotid dropped the pressure from 116/76 to 64/52. During the next 30 minutes it fluctuated between 61/49 and 85/61. The pressures after occlusion of the external carotid as well did not change, giving evidence that no retrograde flow via this vessel was responsible for the fluctuations.

Spontaneous drops which are equally striking and of graver import than the rises can also occur. Thus in Case 1, the pressure distal to a partially closed tantalum band on the common carotid was 80/65 as compared with a level of 120/85 with the flow free. Fifteen minutes later she had become drowsier and showed contralateral weakness and numbness. The distal pressure had dropped to 38/35. We feared a thrombosis, but complete opening of the tantalum band was followed by an abrupt return to normal of the neurologic status and the distal carotid blood pressure.

It is apparent that there were moderate or marked fluctuations in carotid arterial pressure distal to occlusion within the first hour in roughly half of these patients.

PRESSURES IN THE VERTEBRAL TREE

Acting on the assumption that proximal occlusion of a vertebral artery would diminish the pressure within the corresponding posterior cerebral artery which bore an aneurysm, Falconer[5] ligated the vertebral artery low in the neck. That this procedure will reduce at once the blood flow available to the brain stem and posterior cerebral vessels seems highly likely, and Falconer cited two instances from the literature in which ligation of one vertebral artery was followed by an operative fatality in patients who had massive space-taking lesions in the posterior fossa. It does not follow, however, that

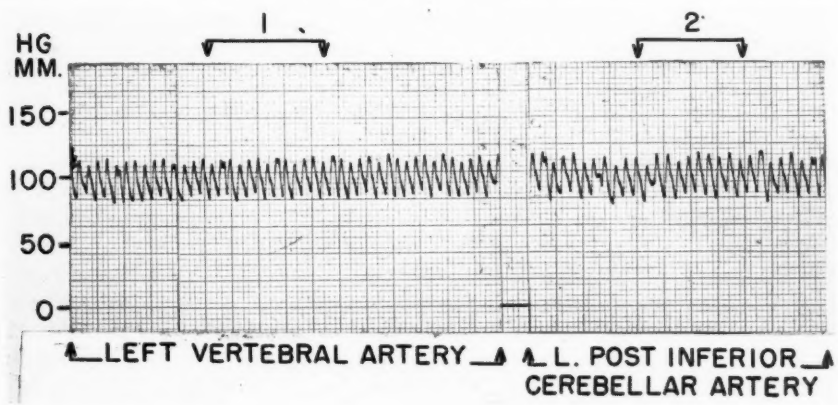

FIG. 3. Complete occlusion of vertebral artery at level of the atlas (1, 2) has no effect distal to the occlusion on pressure in either the vertebral or posterior inferior cerebellar artery.

a reduction in the blood flow in this relatively tiny portion of the cardiovascular system will necessarily be accompanied by a lowering of the arterial pressure. In fact, we should expect the large basilar artery to transmit the pressures from the one open vertebral vessel without loss to both posterior cerebrals and to the opposite closed vertebral artery. We have checked our reasoning during operative exposures in the posterior cranial fossa. The vertebral artery was exposed between the occiput and the atlas and the pressures were measured in both the vertebral and posterior inferior cerebellar arteries after proximal occlusion of the vertebral trunk. Fig. 3 illustrates the utter absence of any change in pressure in these vessels. One might hypothecate that a possible lessening of the blood flow in an artery decreases significantly the pressure within its aneurysm by reducing a presumptive "jet affect" at the aneurysmal opening. We know of no objective measurements to substantiate this, and submit that our findings demonstrate that no fall in pressure, clinically useful in the treatment of aneurysms of the vertebral arterial tree, is produced by occlusion of such artery, assuming that its mate on the opposite side is roughly similar in size.

SUMMARY

(1) Tentatively we find that a substantial acute drop in pressure distal to occlusion of carotid vessels in the neck is maintained for a sufficiently long period to be useful in the treatment of a majority of patients with aneurysms in that carotid system.

(2) We have found no measurable drop in pressure in the vertebral or posterior inferior cerebellar arteries distal to occlusion of one vertebral trunk at the atlas. Hence occlusion of a vertebral artery is not likely to aid in the treatment of aneurysms on vessels supplied by that artery.

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INTRASPINAL NEOPLASMS IN THE CERVICAL REGION*

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THIS presentation summarizes the clinical and pathologic features in 179 cases of intraspinal neoplasms in the cervical region in which surgical treatment was utilized. These cases were encountered at the Mayo Clinic from 1914 through 1950. Sections of each of these neoplasms were re-examined microscopically. Excluded from this series of cases were intracranial and thoracic intraspinal tumors that appeared to involve only secondarily the cervical segments of the spinal cord.

PATHOLOGIC FEATURES

The classification of the neoplasms is listed with their incidence in Table 1. The gliomas consisted of 19 astrocytomas, 17 ependymomas, 3 oligodendrogliomas and 2 unclassified gliomas. Of the vascular tumors there were

TABLE 1

Classification and incidence of 179 intraspinal neoplasms in the cervical region

Classification	Incidence	
	Number	Per cent
Neurilemmoma (neurofibroma)	64	35.8
Meningioma	41	22.9
Glioma	41	22.9
Vascular tumor	15	8.4
Chordoma	4	2.2
Miscellaneous	14	7.8
Total	179	100.0

7 hemangioendotheliomas, 3 hemangiomas, 3 hemangioblastomas and 2 hemangiosarcomas. Among the miscellaneous neoplasms were lipomas, melanomas, lymphomas and sarcomas.

These neoplasms may be classified further according to their location with respect to the spinal canal, the dura mater and the spinal cord itself. Forty-six tumors were intramedullary; although mainly gliomas, they also included 4 vascular tumors, 2 lipomas and a neurilemmoma. Sixty-eight

* Abridgment of thesis submitted by Dr. Webb to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Neurosurgery.

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tumors were intradural but extramedullary; these consisted of meningiomas, neurilemmomas and vascular tumors. Sixteen neoplasms were extradural yet intraspinal. The 49 remaining tumors could be referred to as "dumbbell tumors." They consisted of 38 neoplasms that were both intraspinal and extraspinal, together with 11 other neoplasms that were both intradural and extradural. Furthermore, the intraspinal portion of the former group occasionally was both intradural and extradural. Most dumbbell tumors were neurilemmomas. Among others classed as dumbbell tumors were 4 meningiomas, 4 vascular tumors and 2 melanomas.

The segmental level of these tumors is of some interest. The tumor occasionally extended over several segments of the spinal cord, particularly in the case of intramedullary neoplasms. When this occurred, the segment designated as the level was that of maximal involvement. The 4th through the 7th cervical segments were the sites of 55 per cent of all tumors. Of the meningiomas, however, approximately 60 per cent affected the 1st through the 4th segments of the spinal cord. The 5th or 6th segment was the level of 24 of the 64 neurilemmomas.

Syringomyelia was present in 6 of the 10 cases of intramedullary neoplasm in which necropsy was performed. The 6 cases were as follows: 2 of hemangioendothelioma, 1 of hemangioblastoma, 2 of ependymoma and 1 of astrocytoma. Contrarily, in only 1 of the 15 cases of extramedullary tumor in which necropsy was performed could an associated syringomyelia be demonstrated; in this case, an extensive intraspinal and extraspinal hemangioendothelioma was present.

CLINICAL FEATURES

The entire series consisted of 100 males and 79 females. A girl aged 15 months was the youngest patient, while the oldest was a man aged 76; 8 patients were 12 years of age or younger, yet 4 patients were more than 65 years of age. Of all 179 patients the average age was 38.6. The average age of patients who had meningioma was 42.9, while that of those who had neurilemmoma was 37.6. Patients who had glioma tended to be somewhat younger than the average; indeed, the astrocytomas occurred in patients whose average age was 30.8.

The average duration of symptoms prior to operation for the entire series was 40.9 months. Neurilemmomas produced symptoms 6 months longer, on the average, than meningiomas; the gliomas caused symptoms for an even longer period than the neurilemmomas. Of the common types of tumor the duration of symptoms was longest in the case of ependymomas, namely, 51 months. Symptoms occurred for only several months with hemangiosarcomas, for a somewhat longer period in the case of hemangioblastomas and for a few years when hemangioendotheliomas or hemangiomas were present.

Pain. The commonest first symptom was pain; this occurred in 107 of the cases. The initial pain was unilateral, in the midline or even bilateral

and involved the neck, shoulder or upper extremity in 104 cases. Pain appeared in one or both lower extremities as the initial symptom in 3 other patients, and in these it was diffuse, aching and tended to occur during the night. Histories of pain at some time were obtained from all but 26 patients. The pain experienced by the patients in this study usually corresponded in distribution to the region innervated by the nerve roots at the level of the tumor and it characteristically became intensified when the patient coughed, sneezed, strained and flexed his neck. The pain tended to awaken the patient from sound sleep during the early morning and to be relieved by sitting or walking. Occasionally pain was so mild that the patient mentioned it only incidentally or on questioning. Rarely did pain become so severe that morphine was required for relief; it is interesting that this condition arose in a patient who had an intramedullary glioma.

Generally the pain recurred a few times in the same location during the early stages; then as the illness progressed pain usually increased in intensity, became continuous and extended. Facial pain was experienced by 2 patients after the onset of radicular pain. Indeed, for 6 months prior to operation 1 of these patients had facial pain that closely resembled the pain of trigeminal neuralgia, presumably caused by involvement of the descending nucleus or tract of the trigeminal nerve. The character of the pain in this series of cases was essentially the same regardless of the type of tumor. Although the distribution of pain produced by intramedullary tumors tended to be more diffuse than that associated with extramedullary tumors, still radicular pain was experienced by 14 of the 24 patients who had an intramedullary glioma and who noted pain as the initial symptom.

The distinctive pain, therefore, was valuable because it suggested the presence of an intraspinal tumor and was an important aid in the determination of the level of the tumor; however, the character of the pain was related remotely, if at all, to the type of tumor, and use of the presence or absence of pain was worthless in an attempt to predict the type of tumor.

Sensory Disturbances. Disturbances in sensation exclusive of pain occurred in 156 cases. In 28 of these the changes in sensation constituted the first symptom of the illness. The commonest sensory disturbances were numbness, tingling or a diminished perception of one or more sensory modalities. The initial sensory disturbance occurred in one or both upper extremities in 98 cases, while in 50 cases the first sensory disturbance was noted in one or both lower extremities. Sensory disturbances that involved all extremities at some time occurred in 28 cases. Sensory disturbances that always were confined to one or both upper extremities were observed by 38 patients, whereas 35 patients gave histories of sensory disturbances that always were restricted to one or both lower extremities. The occurrence of a sense of constriction about the abdomen or thorax in 14 patients was misleading in determining the level of the lesion. More than 90 per cent of the neurilemmomas, meningiomas and gliomas were associated with objective sensory disturbances.

The distribution of these objective sensory disturbances permitted determination of the approximate level of the tumor in 127 cases. A well-defined sensory level was obtained in 77 cases. It is important to note, however, that in 3 of these cases the sharp sensory level was extremely misleading, for it occurred at the lower portion of the thorax or the upper part of the abdomen. Objective sensory disturbances were deceptively confined to the lower part of the trunk and the lower extremities in several cases of extramedullary tumor, particularly neurilemmoma and meningioma. But intramedullary tumors rarely produced an objective sensory disturbance whose most cephalad extent was significantly caudad to the level of the tumor. All sensory modalities were impaired in 75 cases. The following modalities of sensation were affected in other cases: pain in 61, temperature in 56, vibration in 36, position in 25 and touch in only 12. Sensory dissociation was observed frequently in this series of cases, and it occurred as commonly with extramedullary tumors as with intramedullary tumors.

Weakness. Weakness was experienced by 165 patients, in 27 of whom it was the initial symptom. It was noted first in an upper extremity in about 60 per cent of these cases, while in approximately 40 per cent it occurred initially in a lower extremity. In cases of meningioma, however, weakness occurred initially in a lower extremity more often than in an upper extremity. Weakness was found in all four extremities of 65 patients. Objective weakness was restricted to one or both upper extremities in 36 cases, of which 15 were cases of glioma. Intramedullary tumors produced weakness that, if not limited to the upper extremities, generally was greater there than in the lower extremities; this was observed especially in patients who had ependymomas. It is significant that objective weakness was confined to one or both lower extremities in 24 cases, of which 10 were cases of meningioma. Weakness was present in all but 3 of the 105 patients who had either neurilemmomas or meningiomas.

Weakness of the diaphragm was demonstrated in 11 cases but this sign proved to be unreliable as an aid in the establishment of the exact level of the tumor. Palatal and pharyngeal weakness was observed in 4 cases in which the tumor involved the upper cervical segments of the spinal cord, and in 2 similar cases weakness of the tongue was evident. The following related signs were associated with weakness: muscular atrophy in 76 cases, fasciculation in 36 and spasticity in 33. Weakness had been regarded incorrectly as a residual of acute poliomyelitis in approximately half the children in this series, particularly during the early phases of the illness when weakness was confined to one extremity.

Reflexes. A disturbance in reflexes was observed in 169 cases. In 163 cases this indicated involvement of the pyramidal tracts, and in 49 of these diminished tendon reflexes were also noted in the upper extremities. This combination was of considerable value in a determination of the level of the tumor. In the 6 other cases the biceps, triceps or radial reflex alone was diminished. It is noteworthy that the tendon reflexes in the upper extremities

were diminished in 67 per cent of the cases of intramedullary tumor; in contrast, this occurred in 20 per cent of cases of neurilemmoma and in only 12 per cent of cases of meningioma. The corneal reflex was diminished on one side in 2 cases in which the tumor involved the upper cervical segments of the spinal cord.

Miscellaneous Signs and Symptoms. Impaired voluntary control of the bowel or bladder or both occurred in 107 patients, but this was of diagnostic significance only in that it indicated disease of the nervous system.

Examination of the neck often revealed helpful signs. Stiff neck was observed in 88 cases and its occurrence suggested the cervical region as the site of the tumor, especially when the tumor otherwise gave the erroneous appearance of being situated at a lower level. Tenderness over the cervical part of the spinal column, occurring in 42 cases, was unrelated to the type of tumor, was often diffuse and, if localized, was frequently misrepresentative of the vertebral level of the tumor; still, this tenderness also drew attention to the cervical region as the site of the lesion. A mass in the neck, which at operation was found to be the extraspinal portion of the tumor, was palpable in 14 cases. When associated with other signs of intraspinal disease, this mass suggested the presence of a dumbbell tumor in the cervical region.

Evidence of increased intracranial pressure was observed in 4 cases of glioma, 3 of meningioma and 3 of vascular neoplasm. Definite papilledema could be measured in 6 cases; blurring of the margins of the disk and distention of the retinal veins occurred in 3 other cases and hydrocephalus occurred in a child. Six of these patients gave histories of recent generalized headache; visual blurring or diplopia troubled 4 of the 10, while frequent vomiting was noted by 2 of the patients.

Functional vascular disturbances, particularly cyanosis, coldness and disturbances in sweating occurred in 24 cases, of which more than half were instances of neurilemmoma. These disturbances usually were observed in an upper extremity of patients who had rather advanced disease and commonly were related to disuse. However, specific disturbances in the vascular system appeared occasionally to be caused by the tumor.

Three patients had Raynaud's phenomenon after the onset of their illness. One of these patients was a meat salesman who had a neurilemmoma that involved the 7th cervical segment of the spinal cord on the right. For 2 years he had observed recurrent episodes of pallor followed by cyanosis of his fifth finger on the right that were induced when he entered a refrigerated room in connection with his work. Intermittent changes in color, which could be classed as Raynaud's phenomenon, involved the hands of the 2 remaining patients.

Horner's syndrome occurred in 35 cases, and was observed in 28 per cent of the cases of intramedullary tumor. When associated with extramedullary tumors the syndrome usually was noted on the same side as that of the tumor. Tumors of the lower cervical segments of the spinal cord in this

series of cases did not produce Horner's syndrome more frequently than did those of higher cervical segments.

Nystagmus was horizontal in 24 cases and vertical or rotary as well as horizontal in 6 other cases. The basis of this sign in these cases is not clear. Barbiturates and other drugs had been administered to several of the patients for relief of pain and sleeplessness. Still, when nystagmus occurred, the tumor usually involved the 1st, 2nd or 3rd cervical segment of the spinal cord.

Intraspinal neoplasms in the cervical region may be responsible for symptoms that one perhaps would attribute to extension of the neoplasm into the medulla; however, in this series of cases, such symptoms often were apparently the effect of vascular changes, edema and displacement of the medulla secondary to the intraspinal neoplasm. One patient was distressed by persistent hiccup. Five patients complained of dysphagia and 4 patients stated that they recently had become unable to speak distinctly.

Ten patients gave histories of dyspnea. This apparently resulted from weakness of the respiratory muscles and the disease usually had become advanced by the time this symptom occurred. A history of dyspnea rarely was obtained from the patients examined during recent years.

LUMBAR PUNCTURE WITH MANOMETRIC STUDIES AND EXAMINATION OF THE CEREBROSPINAL FLUID

The dynamics of the circulation of cerebrospinal fluid were tested by jugular compression after lumbar puncture in 121 of the 179 cases. Complete subarachnoid block was evident in 53 cases; partial subarachnoid block was demonstrated in 40 more cases and in 28 cases a normal response to jugular compression occurred.

The content of protein of the cerebrospinal fluid removed by lumbar puncture was determined quantitatively in 97 cases. Values greater than 40 mg. per 100 cc. were obtained in 83 cases; this increase was especially prominent in cases of complete dynamic block, in which values of several hundred or even a few thousand mg. per 100 cc. were occasionally obtained. The content of protein did not exceed 40 mg. per 100 cc. in 14 cases.

The quantity of protein in the cerebrospinal fluid usually exceeded 40 mg. per 100 cc. when dynamic block was absent; conversely, dynamic block often occurred when the protein content was 40 mg. or less per 100 cc. Both abnormalities were present in 65 cases. But 6 other cases were most unusual in that the content of protein of the cerebrospinal fluid did not exceed 40 mg. per 100 cc. nor could any dynamic block be detected.

The cerebrospinal fluid contained fewer than 5 leukocytes per c. mm. in 79 cases, whereas in 31 cases there were 5 or more leukocytes per c. mm. The number of leukocytes rarely exceeded 10 and counts of 20 to 30 were obtained in only 3 cases.

It is noteworthy that in 19 cases lumbar puncture was followed by an increase in intensity of pain, increased weakness or progression of sensory

impairment leading occasionally to the appearance of a definite sensory level.

SUMMARY

The pathologic and clinical features of 179 cases of verified intraspinal neoplasms in the cervical region have been presented. These neoplasms consisted of 64 neurilemmomas, 41 meningiomas, 41 gliomas and 33 miscellaneous tumors. Sixty-eight tumors were intradural, 46 intramedullary and 16 extradural; the remaining 49 lesions could be termed "dumbbell tumors."

Patients who had meningiomas were usually older than those who had neurilemmomas; the latter were usually older than those who had gliomas. The average preoperative duration of symptoms was 40.9 months. Ependymomas, however, produced symptoms for 51 months on the average.

Sixty per cent of the patients experienced distinctive pain as the initial symptom. Pain was valuable in suggesting the presence of an intraspinal tumor and important in a determination of the level of the tumor; the presence or absence of pain and its character were related remotely, if at all, to the type of tumor.

The reflexes were altered in all but 10 cases. The tendon reflexes in the upper extremities were diminished in 67 per cent of the cases of intramedullary tumor, while 20 per cent of neurilemmomas and only 12 per cent of meningiomas were associated with such changes in reflexes. Weakness and sensory disturbances could be demonstrated in the vast majority of patients, of whom an appreciable number had signs deceptively limited to the lower extremities. Stiff neck, tenderness over the cervical region of the spinal column and a palpable mass in the neck were often present and suggested the cervical region as the site of the neoplasm.

Clinical features have been described that ordinarily are not expected to be associated with intraspinal tumors. Such features appear to be related to the anatomic characteristics of the cervical portion of the spinal cord, including its proximity to the intracranial contents.

Lumbar puncture with manometric studies and examination of the cerebrospinal fluid disclosed either a dynamic block or a value for protein of more than 40 mg. per 100 cc. or both in all but 6 of the cases in which these tests were performed.

CAROTID ANGIOGRAPHY WITH UROKON,* USING THE CHAMBERLAIN BI-PLANE STEREOSCOPIC ANGIOGRAPHIC UNIT

REPORT OF ONE HUNDRED CASES

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Since the introduction of cerebral angiography by Moniz,[17] various complications from contrast media have been reported. The percentage varied from 2 per cent to 15 per cent and the degree from skin rash to hematoma of the neck, convulsions, increased neurological deficit, sarcoma and fatality.[1,2,8–11,14–16,20,22] It is apparent that to decrease these reactions one must reduce the amount of the medium injected and select a substance least irritating to the vascular system. It is also desirable that the procedure be done under local anesthesia. We have attempted to approach these requirements.

In 1951 Chamberlain[6,7] devised his bi-plane stereoscopic angiographic unit using two Fairchild rapid-sequence roll-film casettes. This apparatus takes 12 stereoscopic exposures in both anteroposterior and lateral views in a period of 8.4 seconds, giving a total of 24 frames using only one injection of the contrast medium (Fig. 1). We found that 8–10 cc. of any medium gave adequate filling of the carotid system, both in arterial and venous phases. The reproduction of the vascular system in all phases is equal to that in cerebral angiograms taken by other x-ray units (Fig. 2). Since this apparatus takes serial pictures

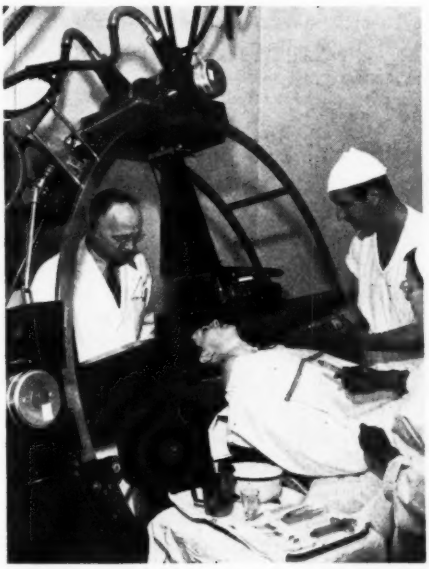

FIG. 1. The Chamberlain bi-plane stereoscopic angiographic unit (see text).

* Urokon Sodium is the sodium salt of 3-acetylamino-2-4-6 triiodobenzoic acid, an organic iodine (65.8% iodine) compound (pH 7 to 7.4) in 30% aqueous solution, manufactured by Mallinckrodt Chemical Company.

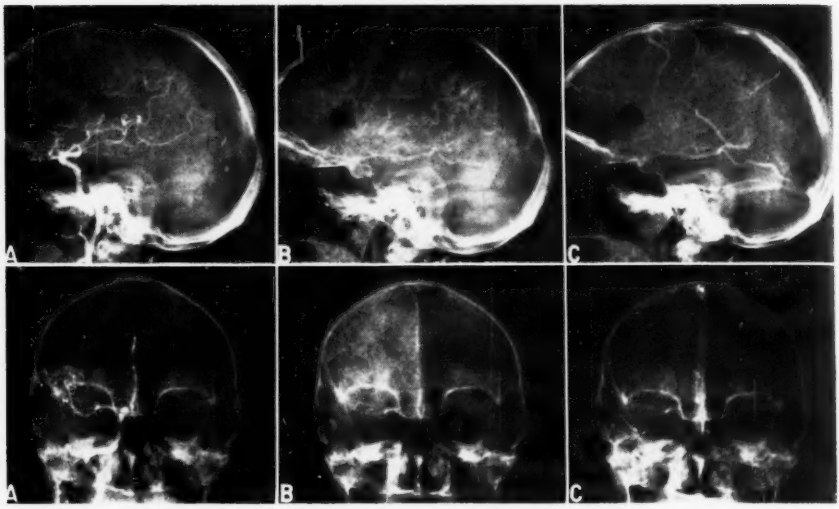

FIG. 2. Normal angiogram with Urokon using the stereoscopic unit. Three characteristic phases were selected from a total of 12 lateral and 12 anteroposterior films obtained from a single injection of 8 cc. in one patient. (A) Arterial phase. (B) Arterial venous phase. (C) Venous phase.

at intervals of seven-tenths of a second, one can determine the cerebral circulation time in its various phases from the angiograms.

Thorotrast has been considered the least irritating of the contrast media. However, its sarcogenic properties are well known.[16] At present, Diodrast (Perabrodil) is widely used in this and foreign countries but it is considered to be irritating.[3–5,10,11,20,22] We have had no experience with Neo-iopax; however, complications from its use have been reported.[12]

This report is based on the use of Urokon Sodium (a relatively new contrast medium) in 100 consecutive cases of cerebral angiography, using the bi-plane stereoscopic angiographic unit. Nesbit and Lapides[19] consider Urokon preferable to Diodrast in urography. Seaman and Schwartz[21] have used 30 per cent Urokon Sodium for cerebral angiography in 36 patients. They reported minor reactions in 7 patients (17.5 per cent), and major reactions in 3 patients (7.5 per cent). However they stated, "A causal relationship to the administration of Urokon is extremely doubtful in the latter group."

"The visible flush of the skin, momentary muscular stiffening and transient pupillary dilatation that invariably accompanied the intra-arterial injection of other radio-opaque materials were absent when Urokon was used."

They concluded that Urokon was a promising radio-opaque medium and warranted further extensive clinical trials.

The manufacturers of Urokon stated that Gass[13] has used the material

in 62 cases and found only 2 per cent neurologic reactions. They also stated that: "Pharmacological studies by Moore[18] have indicated that Urokon Sodium 30 per cent compares favorably with Diodrast 35 per cent in rabbits, using a staining technique as a means of determining blood-brain-barrier breakdown." Bloor[3] found that 1.6 cc. Urokon 30 per cent had a slightly more irritating effect than 1.6 cc. Diodrast 35 per cent when he used EEG tracings and staining technique as criteria. This was found in only 1 animal. In 2 other animals in which 1.6 cc. of Urokon 30 per cent was used, the irritating effect was identical to that from 1.6 cc. of 35 per cent Diodrast; when 3.2 cc. of each medium was compared the Diodrast appeared slightly more irritating.

Our series of 100 carotid angiograms was done on 72 patients. There were 23 cases of tumors, 26 vascular lesions, 3 brain abscesses, 9 cases of convulsive state, and 11 miscellaneous conditions.

Of the 39 carotid angiograms done on the 26 patients with cerebral vascular lesions, 16 angiograms were done on 12 patients with thrombotic lesions; 19 on 10 patients with acute subarachnoid hemorrhage (5 aneurysms; 5 intracerebral hematomas), 2 on patients with aneurysms without hemorrhage, and 2 on patients with a history of old subarachnoid hemorrhage. In this vascular group 7 angiograms were done on patients in coma, 12 on patients whose sensorium was cloudy and confused, and 20 on those with clear sensorium. In this group of vascular lesions 27 angiograms were done on patients with a definite neurologic deficit varying from hemiparesis to hemiplegia. Twenty-three bilateral angiograms were done and of these 14 were done at one sitting.

TECHNIQUE

All conscious patients who do not have increased intracranial pressure receive 100 mg. of Nembutal 1½ hours prior to the procedure; and Demoral 100 mg. and atropine 0.4 mg. 1 hour prior to the procedure. In 60 per cent of the cases 100 mg. of Papaverine were given 1 hour before the study. However, the effect of this drug could not be evaluated. Local anesthesia is used routinely, and pentothal sodium is given intravenously if the patient is uncooperative.

The percutaneous method was satisfactory in all but 3 cases. In 1 of the latter, angiography was done by the open method because of inadequate filling of the carotid system by a previous percutaneous procedure. In the other 2 cases explorations were made to verify the diagnosis of occlusion of the internal carotid artery in the neck suggested by the percutaneous angiograms. In both of these cases the open method verified the occlusion.

The injection of the contrast medium starts immediately after the first exposure has been taken so that the first film shows no dye. This synchronization is important for the calculation of circulation time. The dye is injected as rapidly as possible and it usually takes 2 seconds to complete the injection. If extravasation of the dye is suspected a repeat injection is done. The Fairchild unit presents a disadvantage here for one is unable to determine the result of the angiography until a half hour after the completion of the injection because removal of the film requires movement

of the patient away from the x-ray unit and development of the film takes at least 20 minutes.

Pressure is applied to the neck for at least 5 minutes after the procedure and an ice collar is used for 24 hours. In our series there was not a single incident of hematoma in the neck serious enough to cause any complications. The entire procedure usually takes 5–10 minutes.

Sensitivity to the Urokon is usually tested by the intravenous injection of 1–2 cc. of 30 per cent Urokon 5 minutes prior to the actual procedure.* Eye tests have been done in 50 per cent of the cases. We did not encounter any sensitivity to Urokon.

COMPLICATIONS

In our series 96 consecutive carotid angiograms were done without a single complication such as skin reaction, local complication in the neck, increase in or newly developed neurologic deficit, or fatality. The 97th cerebral angiogram presented the only complication in the entire series.

Case Report. J.D., a male aged 76, was admitted with severe pain over the right temporal region and cheek. Four years previously he had had a cerebrovascular accident resulting in spastic paralysis of the left side of the body and the persistent right facial pain.

Percutaneous right carotid angiography was done under local anesthesia to exclude aneurysm. Ten cc. of Urokon were used, the preliminary intravenous sensitivity test having given negative findings. Four minutes after completion of the angiogram, the patient was asked to move to the stretcher. He sat up and said, "I'm going to die." Immediately following this he became unconscious and rigidity of the neck and upper extremities developed. He regained consciousness in 2 minutes. Heart sounds were indistinct. B.P. dropped to 80/60 and he complained of precordial pain. However, he recovered in 10 minutes without any sequelae. Electrocardiogram taken 30 minutes after the seizure indicated mild cardiac damage, probably old. The carotid angiogram was negative except that the arterial phase was excessively prolonged to 4.5 seconds, indicating impeded blood flow which could be attributed either to arteriosclerosis or to arterial spasm from irritation caused by the contrast medium.

The transient unconsciousness in this case could well have been caused by coronary spasm or insufficiency. However, the fact that there was an impeded blood flow or spasm in the arterial phase indicated definite disturbance of the cerebral circulation. One has to consider this as a case in which there was a complication from the Urokon cerebral angiography.

COMMENT

In our series of 100 consecutive carotid angiograms using 8 to 10 cc. Urokon 30 per cent for each angiogram, only 1 serious but transitory complication occurred. This is considerably less than the complications that we observed when using Diodrast 35 per cent. However, the bi-plane stereoscopic unit was not developed when the Diodrast was used and

* The patients are questioned concerning any history of allergy, particularly asthma, hay fever, hives or reactions to serum.

multiple injections, using as high as 20–30 cc. of the contrast medium, were frequently necessary for each patient. This marked difference in dosage may be an important factor in the difference in complications.

However, volume for volume Diodrast 35 per cent causes more facial grimacing, eye and head pain than Urokon and usually requires a general anesthetic for satisfactory sequence angiograms; with Urokon 30 per cent we were able to do 50 per cent of our studies under local anesthesia.

Thorotrast, discounting its sarcogenic properties, is the least irritating to the vascular endothelium and is the medium of choice in patients over 50 years of age and in those suspected of having occlusive cerebral vascular disease.

CONCLUSIONS

1. The Chamberlain bi-plane stereoscopic angiographic unit has proven valuable in cerebral angiography for the following reasons: (a) It permits the use of small amounts of contrast media for an entire study (8–10 cc.). b) The entire arterial capillary and venous phase is routinely visualized. c) The films are viewed by stereoscopy.

2. Urokon 30 per cent compares favorably with Diodrast 35 per cent in cerebral angiography. However, we noticed milder subjective complaints experienced by the patient during and after injection of Urokon 30 per cent.

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THE SURGICAL MANAGEMENT OF CEREBRITIS COMPLICATING PENETRATING WOUNDS OF THE BRAIN*

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ONE of the most disheartening complications of penetrating wounds of the brain is the development of cerebritis which is commonly accompanied by formation of a cerebral fungus. Experiences in World War II and in the Korean War have shown that earliest possible definitive neurosurgical care of patients with penetrating wounds of the brain should be considered the largest single factor in the prevention of such cerebritis. The employment in Korea of mobile neurosurgical teams close to the line was associated with a reduction of meningocerebral infection from 41 per cent to less than 1 per cent.

Cerebritis as a sequel of penetrating wounds of the brain may be induced by delayed surgical intervention, or by the retention of debris, devitalized tissue and bone fragments. Improper wound closure or faulty healing may then lead to the formation of a cerebral fungus.[1,2,3,7,9,10] Though there are many proposed regimens for handling cerebral fungi,[4,6,8,10,11] the management of frank cerebritis has seldom been approached surgically. It is the purpose of this communication to propose a method of "open" surgical treatment of fulminating, fungating cerebritis which is based on the experiences of one of us in the Okinawa Campaign (1945) and on joint experiences in the Korean conflict. The employment of this method has been associated with a reduction of mortality and morbidity and has been an essential factor in lessening the ultimate neurological deficit.

METHOD OF TREATMENT

Prior to operation antibiotics were administered in accordance with culture and sensitivity studies. Such therapy is maintained for 10 days after closure or until all evidence of infection has subsided. The empirical administration of penicillin 600,000 units b.i.d. and of streptomycin 1 gm.

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The patients in the cases on which this study is based were cared for in the neurosurgical installations of the Far East Command by Edward J. Bishop, Captain, M.C., USAR; Robert A. Clark, Lieutenant (s.g.), M.C., USNR; Philip R. Dodge, Captain, M.C., USA; Gordon T. Wannamaker, Major, M.C., USA, and by the authors.

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b.i.d. has been found advantageous whenever facilities for culture and sensitivity determinations are not available.

1. *Initial Resection.* At operation radical extracerebral debridement with wide resection of the involved portions of the scalp, bone and dura mater is performed. This leads to complete exposure of the fungating mass of necrotic, shaggy cerebrum which is usually surrounded by tense, edematous, but apparently viable brain. The entire fungating mass, or area of cerebritis, is then resected until healthy though sometimes still edematous brain tissue is encountered. Digital palpation will help to determine the tissue consistency and to assure removal of all intracerebral bone fragments. Meningocortical adhesions around the periphery of the wound are divided and resected, allowing the brain to retract within the cranium. The danger of destroying natural barriers to the passage of infection is greatly outweighed by the advantage of direct examination of the subdural space for hematoma or hydroma. Retention in the subdural space of a hematoma, which may also harbor infectious organisms, may be conducive to the continued herniation of cerebral tissue. Upon completion of the initial resection, lumbar puncture is performed to facilitate further retraction of the cerebrum. Withdrawal of 60 to 80 cc. of fluid is usually sufficient to establish a desirable degree of retraction.

The new brain surface is then thoroughly irrigated with saline solution and covered with a square of pure, fine-mesh, autoclaved silk cloth soaked in saline or penicillin solution (5000 units/cc.). The moist, non-irritating piece of silk is covered with strips of vaseline gauze. To protect the brain surface further, gauze fluffs are incorporated in the dressing which is held together by a 5-yard gauze roll.

2. *Interval Care.* The wound is redressed, inspected and irrigated 4 hours after the initial resection and again 8 hours later. The frequency of dressings thereafter is determined by the appearance of the new cerebral surface. So as to keep this surface free from any appreciable accumulation of the products of progressing necrosis, it is usually necessary to repeat the irrigations every 8 to 12 hours.

Each dressing is carried out with the same precautions as those used in the operating room. The wound is exposed, the scalp edges are cleaned with merthiolate and the entire field is draped with sterile towels. Reshaving may be necessary every 2 to 3 days. The surface of the brain is thoroughly irrigated with a continuous stream of saline from an aseptic syringe. Often, 8 to 12 hours after the initial resection, the brain may begin to protrude above the surface of the skull, forming a true cerebral fungus. This is not disastrous, the causative edema being less damaging if the brain is allowed to expand than if it is confined within the cranium by some artificial means. Excessive herniation may have to be controlled by lumbar punctures judiciously repeated whenever the progression of herniation is rapid.* In

* Continuous drainage by means of a polyethylene catheter inserted into the lumbar space may be a practical way of keeping the intracranial pressure low. The employment of a drainage bottle set at an appropriate level makes it possible to predetermine the intracranial pressure.

such cases it may be necessary to separate the scalp edges bluntly from the underlying cortical surface and to irrigate the subdural space.

Duration of the open treatment depends on the appearance of the cerebral surface and on the clinical response of the patient. As infection subsides and debris and exudate are washed away, the surface of the brain assumes a pinkish-gray appearance and takes on a firm consistency; this is usually accompanied by cessation of progressive fungation, indicating that the infection has subsided and that it may be safe to perform secondary closure.

3. *Final Closure.* The patient is prepared for secondary craniotomy. The edges of all layers of the scalp are freshened. The cerebral surface is cleanly resected by sharp dissection, employing silver clips for hemostasis. The dural edges are freed from the cortex and the dural defect is repaired with a fascia lata graft which is secured with interrupted sutures of fine silk. The scalp is closed in layers without tension, using whatever plastic procedure may be feasible in the individual case. Cranioplasty is deferred for at least 12 months.

REPRESENTATIVE CASES

Case 1. An 18-year-old American soldier sustained a penetrating missile wound of the left frontal lobe of the brain on Oct. 9, 1950 while in combat in Korea. Three days later, at a Station Hospital, the wound was debrided and dural closure effected.

Examination. On admission to the Neurosurgical Center at Tokyo Army Hospital on Oct. 13, 1950, the patient was stuporous and febrile. Positive neurological findings were mild papilledema, right facial weakness, mild weakness of the right arm and leg, and hyperactive deep tendon reflexes on the right. The neck was moderately stiff. Inspection of the sutured laceration in the left frontal area revealed a small amount of purulent discharge.

Conservative treatment with irrigation of the wound was of no avail and it was thought that there existed an underlying abscess or cerebritis. X-ray studies did not reveal any retained bone or metallic fragments.

1st Operation. The old wound was reopened, revealing a tight, bulging dural graft with suppurative material exuding from beneath its edges. The surface of the brain was shaggy, necrotic, edematous and grossly infected. The entire area of cerebritis was resected. A direct communication with the anterior horn of the left lateral ventricle was noted, and a piece of gelfoam was inserted in the opening. The wound was left open, dressed with saline-soaked silk and irrigated at frequent intervals as described above.

Course. Herniation of the brain was controlled with lumbar puncture. Ten days later the wound appeared clean, the resected bed was firm and there had been no progressive herniation of the brain after a 24-hour period without lumbar puncture. Simultaneously with the decrease in rate of protrusion of the brain surface, there was marked clearing of the patient's mental status which in the early phases of his treatment had been one of confusion and extreme irritability.

2nd Operation. The presenting surface of the brain was further resected and fascial closure was effected.

Course. Postoperatively, a minimal separation of the scalp in the center of the wound was noted. This healed spontaneously without recurrence of infection. One month later the patient was evacuated to the Zone of Interior. The scalp was well

healed and showed normal pulsations. The neurological deficit that was noted on admission was decreased slightly.

A follow-up letter, received 2 years after the initial injury, reveals that this patient does not have any residual neurological deficit. He has had two epileptic seizures. He also complains of intermittent headaches.

Case 2. An 18-year-old American soldier sustained a penetrating missile wound of the left parietal lobe of the brain on Sept. 15, 1950 while in combat in Korea. The record revealed that a craniectomy had been performed at a forward surgical installation with "evacuation of clots, debridement of cortex and removal of depressed bone fragments." Two days later, at a Station Hospital, the wound was revised and the dural defect was closed.

Examination. On admission to the Neurosurgical Center at Tokyo Army Hospital 7 days later, the patient appeared lethargic, but could be roused. He had a mixed aphasia. There was marked hemiparesis on the right with hyperactive deep tendon reflexes in the right arm and leg. X-ray studies of the skull revealed numerous retained bone fragments within the cerebral substance.

1st Operation. The patient was subjected to reoperation with removal of an extensive subdural hematoma, retained bone fragments and frank pus at the depth of the missile tract.

Course. Seven days later the wound began to separate. After 11 days a large cerebral fungus presented on the surface.

2nd Operation. The fungus and the surrounding cerebritic cortex were resected.

Course. Open therapy was instituted. Three days later, the surface of the brain appeared clean and had taken on a firm consistency. There was no increasing protrusion of brain substance.

3rd Operation. The presenting cerebral surface was again resected and the dural defect was closed with a fascia lata graft. The scalp was closed without drainage.

Course. Postoperatively the patient fared well except for slight separation of the skin edges without evidence of infection. This healed spontaneously. At time of evacuation to the Zone of Interior 2 months later, the scalp was well healed. The patient had regained considerable motor power on the right side. A right upper quadrantal field defect persisted. There was minimal residual dysphasia.

A follow-up letter, received 26 months after the initial injury, reveals that there has been complete recovery from the neurological deficit. The patient is on phenobarbital medication, but has had one seizure. He also reported that cranioplasty had been postponed for 15 months because of intermittent drainage from the superficial layers of the scalp. There has been no infection since then. Following discharge from the Army in October 1952, the patient has been employed in the Vehicle Storage Division of an Air Force Base.

RESULTS

The early results of this open method of treatment of fulminating, fungating cerebritis have been encouraging (Table 1). In 17 cases of cerebritis complicating penetrating wounds of the brain, there were 3 deaths. Two patients died after closure (No. 8 and No. 16) while 1 died during open therapy (No. 5). One of these 3 patients (No. 16) had demonstrated an extremely extensive neurological deficit on admission, suggesting decerebration. This patient had had a complete transection of the sagittal sinus just

TABLE 1

Pt. No.	Wound Location	Ventricle Involved	Duration Open Treatment	Meningitis After Treatment	Superficial Infection After Treatment	Results	
						Infection	Neurological
1.	Rt. parietal	Yes	48 days	No	No	Cured	Imp.
2.	Rt. parietal	Yes	36 days	No	Yes	Cured	Imp.
3.*	Lt. frontal	Yes	10 days	No	Yes	Cured	Imp.
4.	Lt. frontotemporo-parietal	Yes	82 days	No	Yes	Cured	Imp.
5.	Rt. temporoparietal	Yes	—	—	—	Death	
6.	Lt. temporoparieto-occipital	No	9 days	No	Yes	Cured	Imp.
7.	Rt. frontal	No	30 days	No	No	Cured	Imp.
8.	Rt. parietal	Yes	12 days	Yes	Yes	Death	
9.	Rt. frontoparietal	Yes	6 days	No	Yes	Cured	Imp.
10.	Rt. parietal	Yes	7 days	Yes	Yes	Cured	Imp.
11.†	Lt. parietal	No	3 days	No	Yes	Cured	Imp.
12.	Rt. occipitoparietal	No	8 days	No	Yes	Cured	Imp.
13.	Rt. frontoparietal	Yes	3 days	No	Yes	Cured	Imp.
14.	Rt. occipitoparietal	Yes	68 days	No	No	Cured	Imp.
15.	Lt. parietal	No	33 days	No	No	Cured	Imp.
16.	Bi-occipital	Yes	4 days	?Yes	No	Death	
17.	Rt. frontal	No	6 days	No	No	Cured	Imp.

* Representative Case 1 (see text).
† Representaive Case 2 (see text).

proximal to the torcular Herophili with thrombosis of the ends of the proximal and distal segments. The fungus bed appeared to be clean at time of closure and death may not have to be interpreted in this case as a failure of the proposed method.

In patient No. 8 death was preceded by hypothermia, hypotension, bradycardia, and gastric distention suggesting progressive cerebritis involving the hypothalamus. In case No. 5 death was caused by massive cerebritis involving the entire right cerebral hemisphere, associated with ventriculitis. The latter was present at time of institution of open therapy.

The remaining 14 patients were evacuated to the Zone of Interior with healed wounds and without evidence of residual superficial or central nervous system infection. In 10 patients there was persistent superficial scalp infection for a short time after closure, but these had all cleared prior to evacuation. Three patients showed transient clinical meningitis after closure. This responded with complete subsidence to antibiotic therapy. In 1 instance (No. 1) closure was performed too early and open therapy had to be reinstituted before the desired result could be obtained. In no case was there evidence of abscess formation prior to evacuation. There was no increase in the neurological deficit in any of the patients, but all 14 showed definite improvement in the neurological status following completion of open therapy.

DISCUSSION

The method of choice in the management of penetrating missile wounds of the brain is earliest possible definitive neurosurgical intervention with radical debridement, removal of all devitalized tissue and primary closure of the dura mater and scalp. The early phase of the Korean War presented the unfortunate opportunity to see patients with fulminating, fungating cerebritis as sequel of untreated, or inadequately treated missile wounds of the brain. Although all patients in this series had received adequate dosages of antibiotics from the very beginning, cerebritis developed in association with the prolonged retention within the cranial cavity of devitalized tissue, clots and bone fragments. The marked increase in the neurological deficit and the high mortality in those who were subjected to surgical resection of the area of cerebritis and primary closure, led us to adopt the "open" method of treatment which is described in this communication. Its employment, however, has been reserved for previously untreated missile wounds with fulminating, fungating cerebritis and for those patients in whom such cerebritis had developed following inadequate debridement and closure.

Of the 15 patients in the present series who had been operated on prior to institution of open therapy, 13 presented an infected fungus or a draining sinus which led through small openings in the scalp and dura mater to a large underlying area of cerebritis. The remaining 2 were operated on because of bulging flap and other signs of increased intracranial pressure. When the suspected cerebritis was found, it was resected and subjected to open therapy.

Two patients in the series had accompanying lacerations of the sagittal sinus. In 1 (No. 16) the laceration was anatomically complete with thrombosis of the distal and proximal openings into the sinus. In the other (No. 13) there was laceration of the sinus with thrombosis proximal to the tear. The thrombus was evacuated and the laceration was repaired by primary suture and gelfoam.

The presence of ventricular penetration may complicate an existing cerebritis or fungus, but should not offer a deterrent to the institution of open therapy. Ten patients in this series presented ventricular perforations at the time of the first operation at the Neurosurgical Center at the Tokyo Army Hospital.

The duration of open treatment varied from 3 to 82 days with an overall average of 24 days. Though early closure is most desirable, prolonged open therapy is felt to be justified by ultimate cure of an otherwise hopeless situation.

Definitive resection at time of creation of the open wound is advantageous though not always feasible. In some instances further demarcation of non-viable brain necessitates interval resection. In this series 7 patients required from one to three interval resections. In 4 patients, from one to four minor procedures for scalp closure were necessitated by failure of scalp healing without recurrence of fungus or cerebritis. In all patients except in

the one who died during open therapy (No. 5) closure was completed prior to evacuation to the Zone of Interior.

SUMMARY

1. A method of open surgical treatment of fulminating, fungating cerebritis is proposed.

2. Two representative cases are presented in detail and the results of 17 patients so treated are discussed.

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USE OF FROZEN CRANIAL BONE FLAPS FOR AUTOGENOUS AND HOMOLOGOUS GRAFTS

IN CRANIOPLASTY AND SPINAL INTERBODY FUSION

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OCCASIONALLY the neurosurgeon finds it necessary in the course of a craniotomy for various conditions to leave a wide decompression, and therefore must sacrifice all or a part of a cranial bone flap. Under such circumstances he must either discard the bone or find a suitable method of preserving it. If suitably preserved it may be replaced at a later date, thus avoiding the use of artificial materials or other autogenous or homologous bone to fill in the cranial defect. It is the purpose of this paper to report my experience with freezing of such cranial bone flaps and later replacing them and following their course for a period of years. Attention will also be called to the value of preserving cranial bone in this manner for use in homologous cranioplasty in depressed skull fractures, after removal of tumors of the skull, and in other situations where tantalum, plastics, autogenous or homologous bone might be used for cranioplasty.

Although little has been recorded concerning the use of cranial bone preserved by freezing, it has become apparent from conversation with many

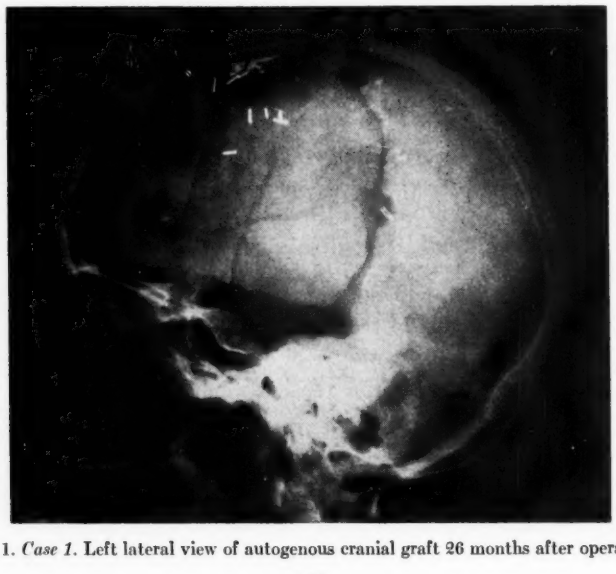

FIG. 1. *Case 1.* Left lateral view of autogenous cranial graft 26 months after operation.

neurosurgeons that such autogenous and homologous grafts are being used not infrequently. It therefore seems advisable to record these experiences.

The writer's experience began in January 1950, as a matter of necessity for decompressive purposes after the removal of a left frontal parasagittal meningioma when acute edema developed in the left frontal lobe. Rather than preserve the bone flap in alcohol, merthiolate, or other antiseptics, freezing was chosen as the method of preservation. The bone flap was wrapped in sterile towels, put in a sterile pan, and again wrapped in sterile towels. A few hours later it was placed in a "deep freeze" at the writer's home. The bone lay in the deep freeze at −10°F. (−23.3°C.) for 16 days. Three hours after removing the bone flap from the freezer it was aseptically inspected at the operating table and found to be in the same condition as when it was removed from the skull. It was then replaced to its former site as an autogenous graft. The wound healed per primam with x-ray evidence of a normal take of this frozen preserved autogenous graft. Roentgenograms have been made of this graft at frequent intervals and now after 2 years the graft shows no evidence of serious shrinkage or loss of calcium content (Fig. 1). (X-rays of the graft at 32 months appear the same as in Fig. 1.)

Since this first experience with freezing a bone flap, the writer has used this same procedure in a total of 5 similar cases.* In addition there are on hand several bone flaps that have not been replaced. Thus there has developed a *cranial bone bank* which is proving useful for homologous grafts. Because experience in these cases has been so favorable, a brief review of the use of frozen cranial bone for homologous bone grafts for cranial defects will be detailed as well as their use in a modification of the Cloward type of spinal interbody fusion after removal of a protruded intervertebral disc.

REPORTS OF CASES

GROUP A. AUTOGENOUS CRANIAL GRAFTS

Case 1. Female, aged 43 years. On Jan. 24, 1950, a left frontotemporoparietal craniotomy was performed for the removal of a parasagittal meningioma, angiomatous in type, weighing 200 gm. The bone flap was removed this same date because of severe cerebral edema.

The flap was placed in the deep freezer chamber and on Feb. 9, 1950, was replaced.

Postoperative roentgenograms taken at 13 months and 26 months (Fig. 1) show an excellent take of the graft.

Case 2. Female, aged 43 years. On Mar. 8, 1950, a left parietofrontal craniotomy was performed for the removal of a meningioma. On March 18 subdural and epidural hemorrhages were evacuated. On March 19 the patient was again returned to surgery because of progressive severe cerebral edema with coincident symptoms of severe increased intracranial pressure. On April 10 osteomyelitis of the bone flap

* Since this was submitted for publication 4 more such grafts have proven successful (2 autogenous and 2 homologous).

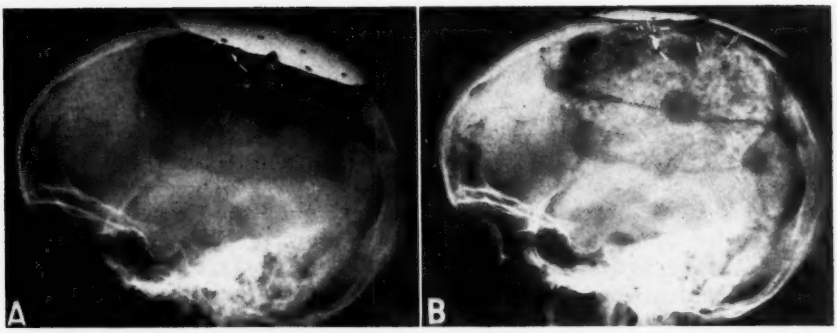

FIG. 2. *Case 2.* (A) X-ray taken 2 months after replacement of autoclaved and frozen (5 months) bone flap, showing beginning absorption of the bone. (B) Showing severe absorption of graft 26 months after replacement.

was disclosed. The bone flap was removed, autoclaved, suitably wrapped, and placed in the deep freezer chamber.

On Sept. 7, 1950, the autoclaved bone flap was replaced.

Postoperative roentgenograms in November 1950 (Fig. 2A) disclosed early absorptive changes in the graft, while those made in November 1952 (Fig. 2B) show very severe absorption of the bone flap.

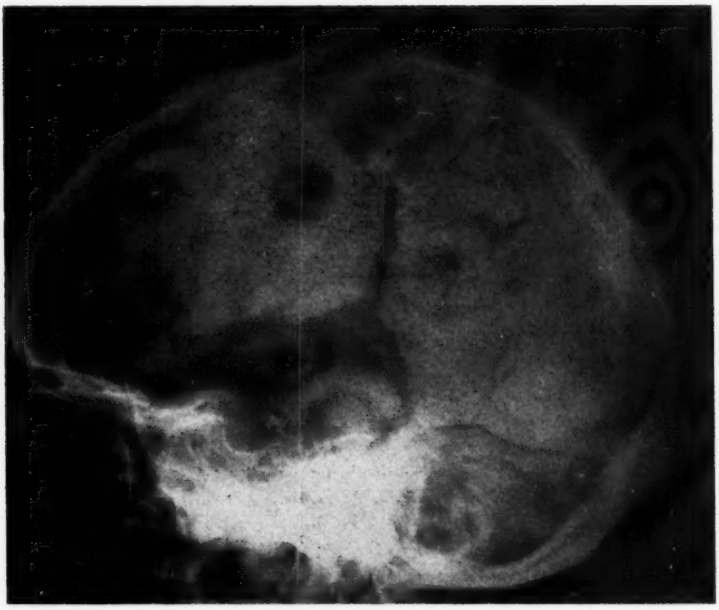

FIG. 3. *Case 3.* Left lateral view showing frozen (24 days) autogenous graft 16 months after operation.

Comment. This is the only infected and subsequently autoclaved and frozen cranial bone flap I have attempted to replace. It is also the only cranial graft that has shown severe absorption. There are probably several reasons why such bone absorbs rather than maintains its normal structure and calcium content. It seems probable that the infection, plus the heat of autoclaving, may have so altered its structure as to prevent revascularization and remineralization.

Case 3. Male, aged 48 years. On Aug. 20, 1950 a left temporofrontal craniotomy was performed for evacuation of a chronic subdural hematoma.

On Aug. 22, 1950, a postoperative subdural and epidural blood clot was removed. Because of acute cerebral edema the bone flap was removed and placed in the deep freezer chamber.

On Aug. 30, 1950, the patient was again returned to surgery. There was a small amount of blood clot with a large accumulation of cerebrospinal fluid in the epidural and subdural spaces. He recovered with moderate neurologic residuals.

Hence, the bone flap was not replaced until Sept. 14, 1950. The postoperative x-rays taken on Jan. 23, 1952 (Fig. 3), 16 months after replacement of the bone flap, show the autogenous graft has taken well without demineralization.

Case 4. Male, aged 68 years. On Sept. 23, 1950 a right temporofrontoparietal craniotomy was carried out for the evacuation of a massive chronic subdural hematoma. On Sept. 24, 1950 the patient was returned to surgery for evacuation of postoperative hemorrhage. On Sept. 26, 1950, because of severe cerebral edema,

FIG. 4. *Case 5.* Lateral view showing frozen (23 days) autogenous cranial bone graft 9 months after replacement.

the bone flap was removed and placed in the deep freezer chamber. He made an uneventful recovery.

On Nov. 9, 1950, the bone flap was replaced. No postoperative x-rays were taken.

The patient expired consequent to a coronary occlusion on Jan. 31, 1951. Autopsy revealed a perfect take of the autogenous skull graft without local complications. There was no evidence of loss of calcium content or absorption of the graft. Unfortunately, no postoperative roentgenograms were available in this case.

Case 5. Male, aged 8 years. On Feb. 19, 1952, a right frontotemporoparietal craniotomy was performed for a probable frontal lobe tumor, but none was found. The brain was under considerable increased tension, the pulse was slow, and the blood pressure was moderately elevated; hence it became apparent that decompression was mandatory. The dura mater was not closed, the brain being protected by a thin film of polyethylene sutured loosely in place. The bone flap was removed and placed in the deep freezer chamber.

Subsequent studies of the tissue removed for biopsy revealed an "eosinophilic encephalitis." [This has been studied by several neuropathologists and no further diagnosis could be made.]

By Mar. 14, 1952, the edema had subsided and the bone flap was replaced. Postoperative x-ray films made in December 1952 (Fig. 4) gave evidence of a satisfactory take of the frozen autogenous graft.

GROUP B. HOMOLOGOUS CRANIAL GRAFTS

(to cranium)

Case 6. Male, aged 45 years. On Sept. 1, 1951 a frontal osteoma was removed. A frozen homologous cranial bone was used as a graft carefully fitted to fill in the area and correct the disfigurement. The bone used had been frozen approximately $2\frac{1}{2}$ months and came from a patient who had succumbed following operative removal of a glioblastoma multiforme that necessitated a decompression.

Postoperative roentgenograms 8 months later show an excellent take of the graft (Fig. 5).

Case 7. Female, aged 39 years. On Apr. 18, 1952 an osteoid-osteoma was removed from the temporofrontal area. A frozen homologous cranial bone graft was used to fill in the area of defect.

A successful take of the graft is shown by roentgenograms taken 8 months later (Fig. 6).

Case 8. Male, aged 9 years. On Mar. 28, 1952 an old small depressed frontal skull fracture was elevated. Although some of the fragments were used to close the defect, more material was needed to fill in the remaining small defect; hence a small piece of frozen cranial bone was chosen. The wound healed satisfactorily.

Roentgenograms taken in November 1952 (7 months later) revealed an excellent take of the graft (Fig. 7) and there was a very good cosmetic result.

Case 9. Male, aged 16 years. One year previously, an eosinophilic granuloma had been removed (by another neurosurgeon), leaving a pulsating defect in the right frontotemporal area. This had worried the patient and he came in for a plastic procedure. On Aug. 11, 1952 a frozen homologous cranial bone graft was wired into the area, followed by primary union of the wound.

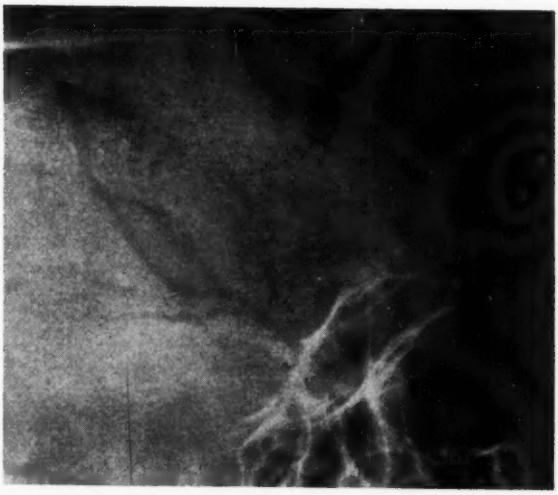

FIG. 5. *Case 6.* Lateral view of frozen homologous cranial graft 8 months after operation.

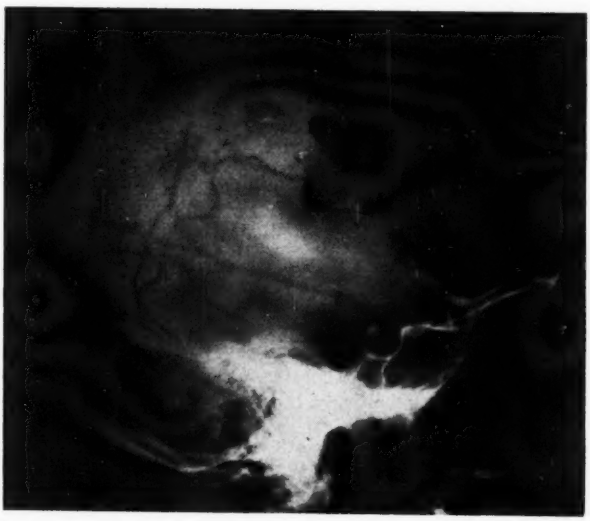

FIG. 6. *Case 7.* Lateral view of frozen homologous cranial graft 8 months after operation.

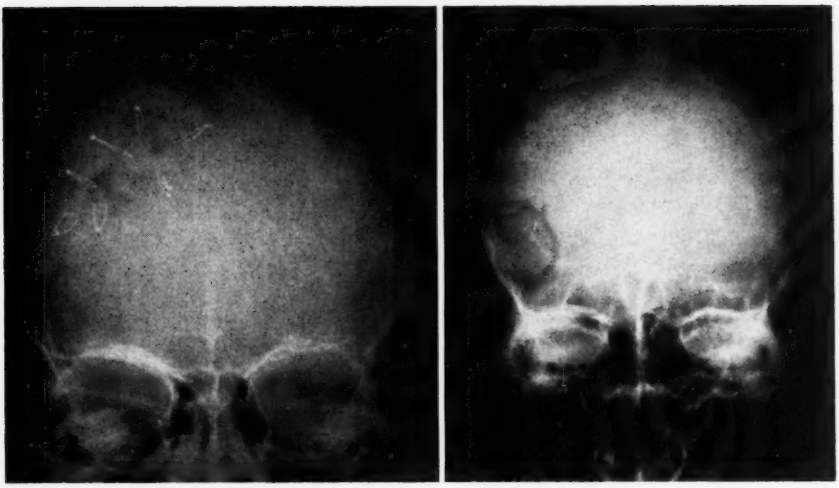

FIG. 7 (*left*). *Case 8.* X-ray of homologous cranial graft 7 months after operation.
FIG. 8 (*right*). *Case 9.* X-ray of homologous cranial graft 4 months after operation.

Postoperative x-rays made in December 1952 (Fig. 8) revealed a good take of the graft, and inspection of the wound disclosed complete disappearance of the defect, leaving a satisfactory cosmetic result.

GROUP C. HOMOLOGOUS CRANIAL GRAFTS

(Spinal fusion—modified Cloward interbody fusion)

The writer has now used multiple discs of frozen homologous cranial bone for fusion of the lumbar vertebral bodies (interbody fusion) after removal of the major portion of the intervertebral disc. The technique employed is a modification of Cloward's[1] operation for the fusion of the vertebral bodies:

A guarded auger drill with pre-set depth stop is used to make the proper size "hole" in the intervertebral area, thus freshening the contiguous edges of the bodies of the vertebrae. Discs of cranial bone are carefully made with a hole saw of the same diameter as the auger drill. These are carefully placed in the intervertebral hole made by the auger. The Cloward vertebral spreader is released and the discs can be seen to seat themselves into the bodies of the vertebrae.

Excellent stabilization of the bodies of the vertebrae can be demonstrated immediately. X-ray evidence of fusion of the vertebral bodies with preservation of the disc space has been demonstrated at a 6-month postoperative period. The technique for "pre-fit dowels" was first carried out by Wiltberger using autogenous bone dowels from the ilium. The writer has chosen to use the harder bone in the hope of maintaining the disc space.*

* More recently a "pre-fit dowel" of iliac cancellous bone has been inserted between two cranial bone discs. This is done to facilitate earlier interbody fusion.

However, the time interval (10 months) is not long enough to evaluate the method. These discs of skull bone can be seen in place at the lumbosacral level in the roentgenogram shown in Fig. 9.

FIG. 9. Lateral view of lumbosacral area showing 3 buttons of homologous cranial bone for interbody fusion in situ 6 months after operation.

DISCUSSION

While it is true that freezing has long been a satisfactory method of preserving bone by orthopedic surgeons, and freezing of soft tissues (nerves, arteries, tendons, fascia, etc.) has been used for other grafting purposes, to our knowledge this is the first report wherein the freezing of cranial bone has been described for autogenous and/or homologous grafting on the skull itself. While it has proven successful in the writer's hands, he is cognizant of the problems peculiar to this and other methods of preserving bone. Attempts to maintain sterility of the skull bone have been done by the following means. First, the bone is immediately wrapped in several sterile towels and as quickly as possible is placed in a freezing chamber where it is "quick-frozen" and then it is not touched again until ready for use. In recent cases the bone has been dipped in or sprinkled with an aqueous solution of penicillin and streptomycin. It is hoped that this will further protect the graft from contamination. Furthermore, it has been the policy to place variable amounts of penicillin and streptomycin solution in the cranial bone-grafted wound just before the closure is completed. The patient is placed on heavy antibiotic therapy for 2 days before and for 5 days after the bone-graft operation.

Whether or not other measures to prevent infection should be tried remains to be seen. So far the precautions as outlined have been sufficient to prevent infection of the graft. Some have suggested that each patient should be placed on heavy antibiotic therapy before each cranial operation. Then if a cranial bone graft is removed it will contain sufficient antibiotic to prevent growth of contaminating organisms. This may be possible but it seems doubtful.

The desirability of having a separate small "deep freeze" type of unit to be used for this bone is immediately apparent. When one has to use a small corner of a large freezer unit used for other purposes, all too frequently various "accidents" will happen to the preciously preserved bone flap.

In preparing "skull bone discs" for a modification of the Cloward[1] type of fusion of the bodies of the vertebrae (interbody fusion) I have chosen to use a "hole saw" and prepare these all at one time from a section of skull

bone. They are packaged in groups of four discs and kept in the freezer unit at −10°F. (−23.3°C.). This procedure has saved a great deal of time and has avoided further trauma and pain to the patient necessitated in the usual procedures.

The writer has not as yet studied the important factors of changes in structure of the cranial bone caused by freezing. The degree of shrinkage of the graft has not been fully ascertained. It has been established that most homologous bone grafts shrink by at least ten per cent. Some of the cranial bone grafts herein studied may have shrunk a little, but it has not been of sufficient degree to be of clinical significance. Studies concerning this matter are under way and will be the subject of a future report.

The course of one autogenous graft (Case 2, Group A) is of special interest for it was an infected bone flap which was autoclaved and then preserved by freezing for 5 months. This is the only graft that has shown serious severe absorption. It would therefore seem inadvisable to preserve and use such a graft. A similar disappointing experience was demonstrated with merthiolate-preserved cranial bone used for a homologous cranial graft.

SUMMARY

A method of preserving cranial bone flaps by rapid freezing for use either as autogenous or homologous grafts in the skull is described. Its successful use in 5 cases of autogenous cranial grafts and 4 of homologous cranial grafts is detailed. Incidental use of homologous grafts of frozen skull bone in the form of small discs for interbody fusions in a modification of the Cloward and Wiltberger type of spinal fusion is recorded. The matters of sterility, contamination, and bone shrinkage are discussed. It is concluded that the freezing of cranial bone when available is a safe, satisfactory, and desirable method. This method allows the use of a normal body tissue for grafting in the skull or spine, rather than using metal and plastic substances.

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DISORDERS OF ELECTROLYTE AND WATER METABOLISM FOLLOWING BRAIN SURGERY

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DESPITE the acknowledged importance of maintaining water and electrolyte balance in surgical patients during the postoperative period, and the many contributions to the subject in the literature pertaining to general surgery, reports concerned with water and electrolyte metabolism in neurosurgical patients have been relatively few. It is the purpose of this report to discuss briefly specific syndromes of disturbance of water and electrolyte balance that are related not only to the physiologic reaction to a neurosurgical procedure, but also, and apparently more specifically, to lesions of the central nervous system that affect neurogenic factors in control of water and electrolyte metabolism. The more general principles of fluid balance have been adequately described elsewhere[11,20] and will not be discussed in this report.

Modern physiologic studies of the role of the brain in water balance originated with the production of urinary diuresis by an experimental lesion of the 4th ventricle.[3,15] The elucidation of the role of lesions of the supraoptico-hypophyseal system in the syndrome of diabetes insipidus established the physiologic relationship of the brain to control of water metabolism.[2,10,24,32] Although neurogenic control of electrolyte metabolism is less well understood, certain facts have been established. One controlling factor in electrolyte balance is the thirst mechanism, which is apparently governed by a physiologic apparatus within the hypothalamus and posterior half of the frontal lobes.[12,33,34] Moreover, Verney[32] has demonstrated the presence of an osmo-receptor in the forebrain which regulates secretion of antidiuretic hormone by responding to the osmolarity of circulating electrolytes. Electrolyte abnormalities secondary to experimental hypothalamic lesions in animals have been recorded by several independent investigators.[16,28]

Several clinical reports have appeared that seem to indicate that electrolyte disturbances may, in certain instances, be secondary to brain lesions. A syndrome of extreme retention of sodium and chloride combined with decreased urinary excretion of these ions associated with cerebral lesions has been independently reported by several investigators.[1,4–7,13,17,19,21,29,35] All reported cases of this syndrome of neurogenic hypernatremia have recently been reviewed by the author.[5] A syndrome that seems to be the exact opposite of neurogenic hypernatremia, that is, excessive salt loss associated with cerebral disease, has been described by Peters *et al.*[22] and confirmed by

others.[14,18,23,25] The recent reports of these two syndromes have re-awakened interest in the role of the central nervous system in the control of electrolyte metabolism. Welt *et al.*[33] have recently stated that "an awareness of the potentialities for disorders in metabolism and excretion of electrolytes and water in association with a variety of primary intracranial abnormalities will increase the frequency of their recognition and provide insights into the mechanisms involved. This must ultimately promote better care for the patient." Since one might justifiably add parenthetically—particularly for the neurosurgical patient—it is the purpose of this paper to review briefly those electrolyte abnormalities most commonly encountered following brain surgery.

NEUROGENIC HYPERNATREMIA

ILLUSTRATIVE CASE REPORTS

Case 1. A 58-year-old housewife, who had had a ventriculocisternal shunt (Torkildsen operation) performed elsewhere 3 years previously, was admitted because of confusion, headaches and blindness.

Examination. Positive neurological findings were complete amaurosis and optic atrophy with superimposed papilledema. Ventriculography demonstrated massive dilatation of both lateral ventricles and the anterior portion of the 3rd ventricle with a round filling defect in the posterior half of the 3rd ventricle.

1st Operation. A suboccipital craniotomy and second ventriculocisternostomy were performed.

Course. The Torkildsen tube functioned only temporarily, and signs of increased intracranial pressure recurred within 2 months.

Metabolic Studies. Throughout this period, from the time of admission, the levels of serum sodium and potassium, plasma chlorides, and carbon-dioxide combining power of the blood were obtained 3 times weekly. All findings were within normal limits.

2nd Operation. Eight weeks after the second ventriculocisternostomy, a right frontal craniotomy was performed, and a transventricular removal of a large brownish, vascular meningioma was accomplished.

Postoperative Course. The patient remained stuporous and became increasingly comatose. The somatic musculature was flaccid. There was marked hypotension (60/40) and hyperthermia (104–105° F.). She died in respiratory failure 1 week following surgery.

Metabolic Studies. After the 2nd operation the serum sodium, which had not risen above 141 mEq/l. in the preceding 8 weeks, rose to 164 mEq/l. by the 3rd postoperative day, and to 169 mEq/l. on the 6th postoperative day, while the plasma chlorides rose to 132 mEq/l. The fluid intake was maintained by gavage feedings at 2500 cc. per day. Urine excretion remained adequate and never fell below 800 cc. in 24 hours. The urine was dilute (sp. gr. 1.004) and acid in reaction during the week prior to death. Urinary excretion of chlorides determined 2 days before death was 80 mEq/l.

Comment. This patient with a known 3rd ventricle tumor was subjected to a control metabolic study for 8 weeks prior to removal of the tumor. All electrolyte studies during this period were within normal limits. Follow-

ing removal of the tumor, however, severe hypernatremia and hyperchloremia developed. The patient was stuporous and the somatic musculature was flaccid.

Case 2. The patient was a 56-year-old colored female.

Operation. Craniotomy was performed with subtotal removal of a craniopharyngioma, which involved the optic chiasm, anterior cerebral arteries and the floor of the 3rd ventricle.

Postoperative Course. The immediate course was relatively smooth but in the ensuing 5 months the patient became progressively lethargic. All four limbs became extremely flaccid during the last 2 months of life. In the last month there was marked edema of the left extremities and left side of the face, and 1 week prior to death the edema became generalized. Death occurred in the 5th postoperative month. Permission for necropsy was denied.

Metabolic Studies. From the time of the 2nd postoperative month, wide abnormalities developed. The plasma chlorides rose to 118 mEq/l., and during the following 2 months, although the patient had a regular hospital diet, the plasma chlorides remained elevated and varied from 103 to 130 mEq/l. There was no evidence of diabetes insipidus, and the urine output varied from 600 to 1600 cc. in 24 hours and contained from 10 to 59 mEq of chlorides per liter.

During a 2-week trial period, the patient received a salt-free diet. Despite this measure the plasma chlorides remained elevated and varied from 112 to 125 mEq/l. During this period of sodium chloride-free diet, urinary excretion of chlorides increased and rose as high as 122 mEq/l. on one occasion, and the serum sodium varied from 139 to 152 mEq/l.

There was no evidence of dehydration during the 5 months of observation. The blood non-protein nitrogen did not rise above 32 mg. per cent, while the hematocrit averaged 41 per cent. The urea clearance test was performed twice and on each occasion was found to be within normal limits.

Comment. In this case marked hyperchloremia and hypochloruria developed following the removal of a craniopharyngioma. The patient was not dehydrated nor did she have diabetes insipidus. She demonstrated flaccidity of the somatic musculature, which had been characteristic of many patients presenting a similar syndrome. Removal of salt from the diet for a period of 2 weeks did not decrease the plasma level of the chlorides.

Cases 1 and 2 are considered representative of patients who have demonstrated a syndrome of marked hypertonicity of the blood following surgical procedures in the region of the sella turcica, hypothalamus and 3rd ventricle. The electrolytic abnormality is characterized by marked retention of sodium and chlorides in the blood with decreased excretion of these ions in the urine. Such hypertonicity of the blood may obtain despite removal of sodium chloride from the diet, suggesting that the hypertonicity is endogenous or metabolic in origin. These patients often become stuporous or actually comatose; the somatic musculature is sometimes flaccid or hypotonic; vascular hypotension often occurs; and hyperthermia is a frequent accompanying sign. The etiology of this syndrome following intracranial surgery is not yet clear. It has been suggested by Allott,[1] Sweet *et al.*,[29] and others[5,6]

that the cerebral lesion may in some way produce the metabolic changes described. In view of the fact that severe hypernatremia has been accompanied by brain damage in almost all cases reported to date, one must consider the possibility that combined neurologic and endocrinologic factors can produce endogenous hypernatremia and hyperchloremia.

In addition to the experiments of Verney,[32] which demonstrated the presence of an osmo-receptor in the brain, other physiologic data suggest that the brain may play a role in the control of electrolyte metabolism. Lewy and Gassmann[16] reported the occurrence of elevated levels of chlorides in the blood of cats following production of experimental lesions in the hypothalamus. More recently, Stevenson, Welt and Orloff[28] reported that the experimental production of lesions in the hypothalamus of rats resulted in the abnormal elevation of sodium in the serum. The role that brain lesions may play in producing this syndrome awaits further observation and evaluation.

We have had little success thus far in the treatment of this electrolyte abnormality once it is recognized. However, we have had some hint of the direction that such treatment should take. There is considerable physiologic evidence that hypertonicity of the extracellular fluid results in the depletion of intracellular potassium.[9] On this basis, and because of the fact that several of our patients with hypernatremia have demonstrated weak hypotonic musculature with electrocardiographic changes suggestive of hypokalemia, parenteral administration of potassium salts is worthy of trial. Moreover, we have been able to effect, in 2 instances, a fall in the level of plasma chlorides by the administration of Pitressin. At the present time, we are investigating the use of cation exchange resins in an attempt to remove sodium from the body in these cases. Such resins are known to result in markedly increased fecal excretion of sodium with a corresponding fall in serum sodium. In addition, the use of mercurial diuretics to promote fluid loss may prove to be of value.

There are several other syndromes of electrolyte disorders following brain surgery that should be noted. These have been previously referred to and therefore will be only briefly summarized here.

SALT WASTING SYNDROME ACCOMPANYING CEREBRAL LESIONS

In some instances, following intracranial surgery, marked excretion of sodium and chlorides in the urine occurs with the abnormal fall of the level of these ions in the blood. This results in stupor, marked weakness, hypotension, and eventual death if unchecked. In many cases in which the syndrome has been recognized, metabolic studies have failed to implicate the pituitary gland or the adrenal cortex as causative factors, despite the fact that this picture is not unlike that seen in some cases of Addison's disease. Peters *et al.*[22] have emphasized the fact that such a syndrome has now been observed following poliomyelitis, hypertensive vascular disease, cranial trauma and after neurosurgical operation. Death is not as inevitable when

this syndrome develops as it has been in most cases of neurogenic hypernatremia. In some cases the tendency to excrete large amounts of salt disappears spontaneously. One should attempt to correct the metabolic imbalance by the administration of sodium chloride. In some instances administration of DOCA has been tried but this is usually accompanied by increased potassium loss and the development of symptoms of hypokalemia. The use of cortisone occasionally offers some benefit in these cases.

ADDISONIAN CRISIS FOLLOWING BRAIN SURGERY

In consideration of neurogenic electrolyte imbalance, as related to brain surgery, one should not lose sight of the fact that many patients subjected to intracranial surgery are in a subclinical state of pituitary deficiency. That this situation is seen in cases of pituitary adenoma is evident. However, it is not as apparent that pituitary deficiency may result from a tumor in the 3rd ventricle, aneurysms of the circle of Willis which evoke pressure upon the contents of the sella turcica, suprasellar tumors, and tumors of the middle third of the sphenoid ridge. In many of these patients with subclinical pituitary deficiency, the stress produced by a major surgical procedure will precipitate a crisis of pituitary and adrenal cortical insufficiency not distinguishable from that seen in Addison's disease. It should be recognized clinically by the low-salt syndrome of nausea, vomiting, hypotension and mental aberrations, the latter varying from changes in the level of consciousness to actual psychotic behavior or coma.[26] The most typical findings are a low blood-sodium value and increased excretion of sodium in the urine.

In order to distinguish this syndrome from the salt-wasting syndrome described above, the ACTH test of Thorn *et al.*[30,31] may be used. This test, involving an eosinopenic reaction to the intramuscular injection of ACTH, is now commonly employed and needs no further elucidation here. A more difficult test, but one which has considerable value, is the water test of Robinson, Kaplan and Power. This demonstrates an inability of a patient with adrenal cortical insufficiency to show a normal diuresis of a water load. It would not be applicable in most instances of Addisonian crisis following a neurosurgical procedure. The treatment of an Addisonian crisis has been described in detail elsewhere[26,27,30] and will be mentioned only briefly. Adrenal cortical extract should be given at once, starting with intravenous administration of 20 cc. of the aqueous extract in one dose, plus 20 cc. given slowly in an intravenous infusion. Five cc. of adrenal cortical extract can then be given intramuscularly. The intravenous administration of aqueous extract is repeated every 2 to 4 hours depending upon the clinical response. Normal saline is given by intravenous infusion and it is usually limited to 2500 cc. in 24 hours. When oral fluids can be taken, intravenous administration is no longer necessary. Desoxycorticosterone, 5 to 10 mg., is given intramuscularly at once and repeated in 12 hours. After the crisis has been managed, this dose is gradually reduced to 1 to 2 mg. given every 6 to 12 hours for 2 to 3 days. Administration of antibacterial agents is always indi-

cated as are general measures to contract shock. The recognition of Addisonian crisis following intracranial surgery is particularly important, and should be suspected in any case in which nausea, vomiting, hypotension and mental changes develop following intracranial surgery.

HYPOKALEMIA

Much has been said recently of the syndrome of low serum potassium following surgical procedures. It is worthy of note that many factors that can produce potassium depletion with its attendant muscular and respiratory complications are present in neurosurgical patients. Among these are the catabolic responses to craniotomy previously described by the writer and coworkers.[8] Acidosis, alkalosis, starvation, and hypertonicity of the extracellular fluid, all of which are not uncommonly seen in neurosurgical patients, may result in hypokalemia and the syndrome of potassium depletion.[11,20] Therefore, one should be particularly interested in the level of serum potassium following intracranial surgery. The administration of potassium parenterally, of course, is the specific treatment for this abnormality.

DISCUSSION

It is not the purpose of this presentation to elaborate upon the clinical and laboratory evidence supporting the role of the central nervous system in regulation of water and electrolyte balance. Rather, it is desired to emphasize two facts. First, there are certain disorders of electrolyte metabolism, such as severe hypernatremia, which appear to occur predominantly in patients with pathologic or surgical lesions of the brain. Secondly, it lies within the province of the neurosurgeon to contribute to a better understanding of neurophysiologic mechanisms involved in the control of water and electrolyte metabolism. As previously stated by Welt *et al.*,[33] "this must ultimately promote better care for the patient."

SUMMARY

Some of the neurogenic factors concerned with the control of water and electrolyte metabolism have been reviewed. The outstanding neurophysiologic mechanisms presently known, appear to be the following: (1) The role of the central nervous system in control of thirst. (2) The supra-optico-hypophyseal mechanism concerned with the release of antidiuretic hormone. (3) The cerebral osmo-receptor mechanism of Verney. Other neurogenic factors doubtlessly exist.

Four syndromes of abnormality of electrolyte metabolism particularly apt to occur postoperatively in neurosurgical patients have been described. These are: (1) Neurogenic hypernatremia and hyperchloremia. (2) Salt-wasting syndrome accompanying cerebral lesions. (3) Postoperative Addisonian crises in cases of chronic adrenal cortical or pituitary insufficiency. (4) Postoperative hypokalemia. The therapy of these syndromes, insofar as known, has been outlined.

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THE RELATIVE ACCURACY OF ELECTROENCEPHALOGRAPHY, AIR STUDIES AND ANGIOGRAPHY

IN A SERIES OF TWO HUNDRED MASS LESIONS*

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IN THE early part of this century the diagnosis of an intracranial mass lesion was made by clinical observations and roentgenographic studies. It was possible to localize a mass lesion in 40 to 60 per cent of patients by clinical studies. Roentgenograms, at first not too dependable, were of help in 15 per cent of cases. Ventriculography and pneumoencephalography were introduced in 1918 and 1919 by Dandy. Cerebral angiography was employed by Moniz in 1927, a procedure used infrequently in this country until the introduction of a percutaneous method of injection and a less toxic solution. Berger described electroencephalography in 1929.

More than two decades of experiences with these procedures have been recorded in the literature. Several observers[2,3,4,5] have compared their diagnostic value. It is the purpose of this paper to compare the relative accuracy of encephalography, ventriculography, angiography and electroencephalography as they were used in the diagnosis of 200 verified, selected, but consecutive cases of mass intracranial lesions in a 4½-year period.

MATERIAL AND METHODS

The cases were selected on the basis of the following criteria: first, the type of lesion was space-occupying; second, the site of the lesion was established by surgery and/or autopsy; and third, the case was studied by several diagnostic methods. The histopathology was determined in all but 18 cases (Table 1).

The 224 electroencephalographic records used in this analysis were produced on an 8-channel, ink-writing, Model 3, Grass electroencephalograph. About 90 per cent of the records were recorded from leads placed according to the method of Gibbs. These records were essentially monopolar in origin, although short "runs" of bipolar leads were present in each. The other 10 per cent of the records were bipolar. The bipolar leads were placed according to a modification of the technique of Jasper. The electroencephalographic localization per se was made exclusively on the basis of the tracing. However, interpretation of the record was made in the light of the history.

The 90 pneumoencephalograms were performed by the removal of

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TABLE 1

I. MASSES OF THE CEREBRAL HEMISPHERES				151
(A) *Neoplasms*			97	
(1) Glioma		77		
Glioblastoma	34			
Unclassified	14			
Astroblastoma	7			
Spongioblastoma	6			
Oligodendroglioma	5			
Astrocytoma	11			
(2) Meningioma		12		
Benign	9			
Malignant	3			
(3) Hemangioma		5		
(4) Metastatic		3		
(B) *Hematomas*			45	
(1) Subdural		37		
Acute	5			
Subacute	3			
Chronic	29			
(2) Intraparenchymal		8		
(C) *Abscesses*			8	
II. MASSES OF THE POSTERIOR FOSSA				30
(A) *Neoplasms*			30	
(1) Glioma		11		
Unclassified	4			
Spongioblastoma	3			
Astroblastoma	1			
Astrocytoma	3			
(2) Metastatic		6		
(3) Medulloblastoma		5		
(4) Meningioma		3		
(5) Neurofibroma		3		
(6) Hemangioma		1		
(7) Neuroblastoma		1		
III. MASSES OF THE MIDLINE BASE				19
(A) *Neoplasms*			19	
(1) Meningioma		9		
(2) Pituitary tumor		5		
(3) Chordoma		2		
(4) Craniopharyngioma		2		
(5) Neurofibroma		1		

cerebrospinal fluid and replacement with room air through the lumbar route. With the exception of cases of massive hydrocephalus, an attempt to replace the entire amount of fluid was made. This usually involved the exchange of 100 to 150 cc. The roentgenograms were made in both vertical and horizontal positions.

The 100 ventriculograms were performed by the replacement of ventricular fluid with room air through two burr openings centered at points 8 cm above the external occipital protuberance and 3 cm. from the midline. All roentgen studies were made in the horizontal position.

The 58 carotid angiograms, with the exception of 3, were performed by the percutaneous method. Thorotrast was used in patients 55 years of age or more, or in those who were sensitive to diodrast.

RESULTS

Electroencephalography. Of the 224 preoperative electroencephalograms, which were recorded in 198 patients, 168 were abnormal. The diagnosis was aided by EEG in 101 cases, the graph indicating the site or cranial fossa of involvement. However, only 93 (47 per cent) of the lesions were localized to a discrete area. When posterior fossa and midline base lesions were excluded, an accuracy of 59 per cent was found (Table 2).

TABLE 2

Electroencephalogram

	Negative	Diffuse	Lateral-izing	Fossa Focus	Exact Focus	False Focus
Masses of the cerebral hemispheres						
Neoplasms 97	8	13	2	0	59	15
Abscesses 9	0	0	0	0	9	0
Hematomas						
Intraparenchymal 8	0	0	0	0	8	0
Subdural 36	2	4	3	0	14	13
Masses of the midline base						
Neoplasms 18	12	0	1	0	3	2
Masses of the posterior fossa						
Neoplasms 30	8	5	0	2	0	15

Accurate localization was made most often in cases of rapidly growing gliomas and the malignant meningiomas over the convexities. These were localized in 66 per cent of the cases.

The growth rate of the mass appeared to influence the frequency of appearance of discrete foci. The slower-growing gliomas of the hemispheres were localized in 62 per cent of the cases. The benign meningiomas involving the convexities and the hemangiomas of the hemispheres could be localized to an exact focus in 44 per cent and 40 per cent respectively.

Three of the 9 meningiomas that occurred at the midline base were localized by EEG because they produced pressure against the frontal lobes with EEG changes.

No exact focus was noted in any case of posterior fossa mass irrespective of the growth rate or predominant side of involvement.

Thirty-nine per cent of the subdural hematomas were localized to an area circumscribed well enough to be confined in a surgical bone flap.

All 8 intraparenchymal hematomas and all 9 of the abscesses were localized to an exact site.

False foci were found in 45 of the 198 cases. Fifty per cent of the masses

of the posterior fossa were falsely localized by the EEG. In the cases of posterior fossa lesions the appearance of false focal disturbances followed no consistent pattern. They appeared in both frontal and occipital leads more frequently than temporal leads. They occurred on either the same or on the opposite side of the tumor. Occasionally, the pattern was suggestive of a thalamic tumor.

Thirty-six per cent of the subdural hematomas were falsely localized by the EEG. Of the 37 hematomas 29 were chronic and existed with no severe associated cerebral damage. The presence of cerebral damage was not a factor causing false foci.

Eleven cases (all mass hemispheric lesions) were studied preoperatively by multiple EEGs. The following observations were noted: (1) Local extension of a discrete focus occurred in 8 of the cases. (2) Extension of a discrete focus across the midline to become a diffuse, random, or symmetrical abnormality occurred in 3 cases. (3) Intracranial hypertension was present in 9 cases with discrete foci which persisted in the presence of an increasing intracranial pressure. (4) In 6 cases there were focal abnormalities so early in the course of the disease that the mass was not evident by pneumoencephalographic or angiographic examination performed at that time.

Pneumoencephalography. Of the 90 pneumoencephalograms employed in the diagnosis of 82 cases, 46 were recorded as abnormal (Table 3). Thirty of the tumors were localized by this method, an accuracy of 36 per cent.

TABLE 3

Pneumoencephalogram

		Negative	Lateral-izing	Fossa Focus	Exact Focus	False Focus	Diagnostic Failures
Masses of the cerebral hemispheres							
Neoplasm	38	6	4	0	15	0	13
Abscess	3	0	0	0	3	0	0
Hematoma							
Intraparenchymal	2	0	0	0	2	0	0
Subdural	17	3	9	0	2	1	2
Masses of the midline base							
Neoplasm	13	4	0	0	8	0	1
Masses of the posterior fossa							
Neoplasm	9	0	0	1	0	1	7

Accurate localization of tumors was made most often in cases of rapidly growing gliomas of the cerebral hemispheres. Forty per cent of these were localizable.

Eleven of the 17 subdural hematomas were lateralized. However, only 2 were localized to an exact site. Two intraparenchymal hematomas and 3 abscesses were localized in all cases.

None of the posterior fossa masses could be localized to an exact site and only 1 was localized to this fossa.

TABLE 4

Ventriculogram

		Negative	Lateralizing	Fossa Focus	Exact Focus	Diagnostic Failures
Masses of the cerebral hemispheres						
Neoplasms	55	2	3	1	47	2
Abscesses	3	0	1	0	2	0
Hematomas						
Intraparenchymal	2	0	0	0	1	1
Subdural	4	0	1	0	3	0
Masses of the midline base						
Neoplasms	3	1	1	0	1	0
Masses of the posterior fossa						
Neoplasms	29	2	0	24	3	0

Eight of the 13 tumors that occurred at the midline base were localized by this method. In most cases this was because of defects in the pattern of the basal cisterns rather than the ventricles.

There were 23 diagnostic failures because of insufficient aeration of the ventricular and subarachnoid spaces. Seven of the 9 pneumoencephalograms performed in cases of posterior fossa tumors, and 13 of the 38 performed in cases of hemispheric tumors were failures. In 11 of the cases successful ventriculography was subsequently performed.

Ventriculography. Of the 100 ventriculograms performed on 96 patients, 4 were diagnostic failures because of insufficient ventricular filling. These were successfully repeated. In 88 of the studies the findings were abnormal and in 86 they were diagnostic. This is an accuracy of 90 per cent.

Of the 29 lesions in the posterior fossa, only 3 were localized to an exact site. However, 24 of these studies localized the lesion to the fossa.

In only 1 of the 3 ventriculograms performed on patients with midline base tumors was the site of the tumor revealed.

TABLE 5

Angiogram

		Negative	Exact Focus	False Focus	Diagnostic Failures
Masses of the cerebral hemisphere					
Neoplasms	37	14	20	1	2
Abscess	1	0	1	0	0
Hematomas					
Intraparenchymal	5	1	4	0	0
Subdurals	6	3	3	0	0
Masses of the midline base					
Neoplasms	8	5	3	0	0

As will be noted in Table 4 it was possible to localize accurately 75 per cent of the hematomas and abscesses by this method.

Angiography. Fifty-seven carotid angiograms were performed on 57 patients. Of the 57 masses 31 were localized to an exact site. Among the neoplasms that occurred in the hemispheres, 57 per cent were accurately localized by this method. These masses occurred in the anterior two-thirds of the hemispheres.

Half of the subdural hematomas (6 cases) and midline base lesions (8 cases) were localized. The intraparenchymal hematomas and abscesses were also localized with a high degree of accuracy (Table 5).

DISCUSSION

No single method is adequate in the localization of all types of intracranial mass lesions. The method of choice will depend not only on the physical status of the patient, but also upon the diagnostician's clinical concept of the site of the lesion.

Electroencephalography may be used in all patients capable of cooperating, but it can be considered only as an adjunct to other diagnostic methods. It must be remembered that projected EEG abnormalities may be falsely interpreted as focal abnormalities and focal abnormalities tend to become generalized with progression of the disease.

Local extension of a discrete focus is probably caused by local growth of the tumor rather than by increased intracranial pressure. Nine of the 11 patients who were studied by multiple EEGs had persistent foci in the presence of a rise in intracranial pressure. A focal abnormality became a general abnormality in 3 cases. This extension of the abnormality may be the result of invasion of midline structures or may be caused by artefact from monopolar lead placement which utilizes ear leads in common.

Electroencephalographic examination of the patient early in the course of his disease is desirable. Not only is it possible to obtain more discrete foci, but in 6 of the 12 cases, an EEG focus was noted when pneumoencephalographic and angiographic findings were negative.

Encephalography is useful in the diagnosis of lesions above the tentorium cerebelli. Masses that occur in the basal cisterns, an area that is not visualized by ventriculography, may be delineated by pneumoencephalography.

Failure of filling of the ventricular system is presumptive evidence of an intracranial mass lesion. The presence of increased intracranial pressure as a cause for these failures was first suggested experimentally by Jüngling[3] and clinically by Bohn,[1] who reported tumors and anesthesia as causes for the rise in intracranial pressure. In our group, although all had mass lesions, none had papilledema at the time of pneumoencephalography and only 2 had general anesthesia.

Ventriculography is the most nearly accurate method available. It is

he method of choice in cases of posterior fossa lesions or when intracranial hypertension is present.

Carotid angiography is most useful in the diagnosis of lesions occurring in the anterior two-thirds of the cerebral hemispheres and those that occur at the midline base (optic and supra-optic region). It is the method of choice in the desperately sick patient with a mass lesion at these sites.

SUMMARY

The relative value of electroencephalography, pneumoencephalography, ventriculography and angiography in the diagnosis of 200 cases of mass lesions is discussed.

Electroencephalography is useful only as an aid to other diagnostic methods.

Pneumoencephalography is valuable in the localization of hemispheric mass lesions and is the procedure of choice with tumors at the midline base.

Ventriculography is the most useful method in the localization of any intracranial mass lesion, having a general accuracy of 90 per cent. It is the only method, with the exception of vertebral angiography, that is helpful in the diagnosis of posterior fossa masses.

Masses of the anterior two-thirds of the hemispheres and about 50 per cent of those that occur at the midline base may be localized by carotid angiography.

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THE TREATMENT OF ANEURYSMS OF THE CIRCLE OF WILLIS

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THE prognosis of spontaneous subarachnoidal hemorrhage caused by rupture of aneurysms on the circle of Willis has been the object of several recent investigations. The results of the more important of these statistics are summarized in Table 1. It is evident from the figures presented that the prognosis of these aneurysms once they have started to bleed is very serious, between 35–50 per cent of the patients dying from the effects of the initial hemorrhage. A considerable proportion of those dying during the initial attack do so within the first 48 hours—according to Dandy's[2] figures, about 50 per cent and among these again about half within the first 24 hours.

Recurrent bleeding occurs in a large number of patients who survive the initial attack. The percentage of recurrent bleeding shows rather wide variations in different statistics, from 20 per cent reported by Hyland[6] up to 50 per cent reported by Hamby.[5] All agree, however, that the mortality attending secondary hemorrhage is even greater than in the initial bleeding, and mortality figures up to 70–80 per cent have been reported (Table 1).

TABLE 1

(*From Erik Ask-Upmark and David Ingvar*[1])

	No. of Patients	Death During First Attack	Recurrences (total)	Recurrences (fatal)	No. of Survivors
Mage	150	52	50	32	66
Dandy	59	30 (+6)	15	12	11
Wolff *et al.*	46	5	24	10	31
Hamby	130	44	46	39	48
Ask-Upmark and Ingvar	138	38	39	23	77
Total	523	169 (+6)	174	114	233

Since the pioneer work of Dandy[2] the intracranial aneurysms have been a subject of great interest to neurosurgeons and numerous important contributions have been made in recent years. It does not seem to have been generally recognized, however, that two quite different problems are involved relating to the treatment of intracranial aneurysms. The first and most

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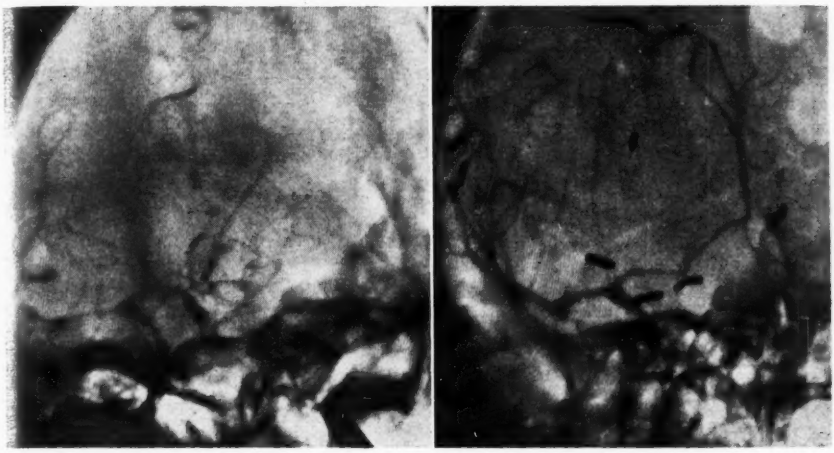

FIGS. 1 and 2. *NSC 705/51.* G. G., female, aged 49 years. (*Left*) Arteriogram 10 days after subarachnoidal hemorrhage shows aneurysm of anterior communicating artery with intense vasospasm in carotid artery and its main branches. (*Right*) Postoperative arteriogram shows clips on neck of the aneurysm, which is no longer filled. Disappearance of vasospasm.

pressing question is what should be done in the way of surgery, if any, to aneurysms in the acute stage of hemorrhage. The second problem, which appears to be much easier of solution, is what should be done to prevent recurrent bleeding. Unfortunately the statistics so far published do not distinguish between these two problems, and the results of various procedures are assessed irrespective of the stage of the aneurysm in relation to leakage. This greatly confuses the issue and could be compared to an attempt to evaluate different methods of treating, for instance, inguinal hernia irrespective of whether the hernia was strangulated or free.

In the stage of acute hemorrhage an aneurysm is a very grave condition entailing great danger to life and to function in case the patient should survive. It may be said in consequence that considerable surgical risk may be justified if thereby the mortality attending the first attack of subarachnoidal hemorrhage can be materially reduced. However the timing of the operation is a factor of such great importance that without information on this point no conclusions at all are possible concerning the result. Among the large number of cases reported in the literature such information is available only in isolated instances. It would appear therefore that a new approach to this problem is indicated.

Before proceeding further some experiences concerning operations in the acute stage may be recalled. We know from the work of Jefferson[7] and Schorstein[11] that carotid ligature shortly after subarachnoidal hemorrhage has occurred is much more dangerous than after the acute effects of hemorrhage have subsided. As will be shown, the experiences of this clinic

indicate that direct attack on the aneurysm by clipping its neck or trapping is also very dangerous in the acute stage. The reasons for this unfavorable reaction are (1) impairment of the cerebral circulation caused by the presence of blood in the subarachnoidal space or clots in the brain substance with consequent rise in the intracranial pressure; and (2) the intense vasoconstriction nearly always present in the cerebral arteries shortly after hemorrhage has occurred and which can be demonstrated in the arteriograms (Figs. 1 and 2). Vasoconstriction may possibly be the more important of these factors since a considerable part of the extravasated blood can be removed if an intracranial approach is made. The intense vasoconstriction frequently induced by leakage of the aneurysm is to be considered as a protective mechanism to prevent further hemorrhage. How long this vasoconstriction persists we do not know, but there are indications that it may persist as long as 2–3 weeks. Possibly relaxation of the vascular tone a week or two after the initial hemorrhage may be responsible for the recurrence of hemorrhage so frequently observed at this time. It is possible that the arterial spasm observed in the arteriograms is caused in part by the contrast medium (diodrast). Falconer[4] observed in one case, in which arteriography was done first with diodrast and the following day with thorotrast, that arterial spasm could be seen only in the diodrast-arteriogram but not in the other. However the arterial spasm seen in the arteriograms can often be confirmed days later by direct inspection of the arteries in the operative field. It seems probable, therefore, that vasoconstriction is a reality in many cases and not an artefact caused by the contrast medium. Also vasoconstriction is almost never observed in arteriograms made in conditions other than aneurysms and occurs in the latter only when there has been recent hemorrhage.

Falconer[4] reported a series of 50 intracranial aneurysms operated on during the first weeks after hemorrhage had occurred. It is not clear from his report how long after onset of bleeding the operation was performed and if there was any correlation between timing of operation and mortality or recovery with or without defects. The operation employed in his cases was ligature of the carotid artery in the neck sometimes supplemented by intracranial trapping, clipping of the neck of the aneurysm or wrapping with hammered muscle. The various procedures employed by him resulted in 33 good recoveries, 9 fatalities and 8 recoveries with more or less serious defects. In relation to the number of cases there are too many variations with regard to the procedure employed and too little is known concerning timing of operation and presence or absence of arterial spasm to permit conclusions other than the rather vague one that mortality and morbidity appear to be approximately the same as would be expected in a similar group treated along conservative lines.

Our own material (Table 2) shows that of 15 patients operated on between a few hours up to 22 days after onset of hemorrhage, 8 died. Good recoveries followed in 6 cases and in 1 case hemiplegia gradually set in the

TABLE 2

Intracranial explorations for intracranial saccular aneurysm in the acute stage

	No. of Patients	Method				Dead
		Lig. of Neck	Wrapping	Trapping	Explor.	
Carotid artery	7	5		2		4
Bifurcation of internal carotid artery	2	2				2
Anterior communicating artery	4	3	1			1
Middle cerebral artery	2	2				1
Total	15	12	1	2		8

day after operation and became permanent. The procedure employed was ligature of the neck of the aneurysm in most cases. In 2 cases operation was performed within a few hours after onset of massive hemorrhage, necessitating in 1 case resection of the frontal lobe to allow access to the aneurysm. In neither of these cases was appreciable vasoconstriction present, and the hemorrhage was so extensive that the patients would probably have died within the first 2 days irrespective of what form of treatment had been employed. In the remaining 6 cases in which fatality occurred pronounced vasospasm was present in 5; in the 6th case, in which no vasospasm was noted at arteriography and operation 2 days after onset of hemorrhage, the patient made a fairly good recovery from the operation but died 2 weeks later of pulmonary gangrene. In the case in which hemiplegia developed after ligature of the neck of an aneurysm on the internal carotid artery, the posterior temporal artery, which could be clearly seen in the first arteriograms was found to be occluded 3 weeks later when a control arteriogram was made. Since this artery was outside the immediate field of operation the cause of thrombosis of this vessel remains unexplained.

Our impression from these experiences is (1) that when massive hemorrhage has occurred, no operation is likely to save the patient, and that as a rule it is useless to operate unless the patient survives the first 2 days, and (2) that the circulation of the brain is profoundly disturbed during the first 2 or 3 weeks following hemorrhage, and this disturbance is the result both of the increase in intracranial pressure, and the vasoconstriction almost invariably present in these cases. Unless some method can be found to eliminate vasoconstriction after the neck of the aneurysm has been clipped, it is probably better to abide by conservative treatment. We are at present experimenting with papaverine and permanent perfusion of the superior cervical ganglion as recommended by Poppen,[10] but whether any or both of these methods will contribute materially to reduce the hazards of early operation cannot yet be determined. It is perhaps also possible that by postponing the operation until such time that the vasoconstriction begins to

subside spontaneously the dangers of the operation may be reduced. This would appear to be about 3 weeks after hemorrhage as found by Ecker and Riemenschneider,[3] a view that is also supported by our own experiences. Only further experiences can tell whether measures aiming at reducing vasospasm or/and judicious timing of operation will give better results. So far our experiences indicate that operations without such measures in the acute state, that is, within the first 3–4 weeks after hemorrhage, result in a mortality that is about the same as if the cases had been left to run their spontaneous course. To the credit side of operations in the acute stage may be added, that if the patient survives clipping of the neck of the aneurysm, he does not run the risk of a second hemorrhage later on.

However, the high mortality, 50 per cent or thereabout, associated with bleeding aneurysms left to run their spontaneous course presents a challenge to neurosurgery of sufficient importance to justify further attempts to deal with these lesions in the acute stage. Whether indirect methods, such as ligature of the carotid artery in the neck, or a direct attack on the aneurysm by clipping its neck should be preferred cannot yet be decided on the basis of available evidence. We incline to the view that clipping of the neck of the aneurysm is the more promising method. If the patient survives this procedure, he is definitely cured of his illness and methods aiming at reducing vasospasm can be applied without hesitation. Whether ligature of the carotid artery in the neck does prevent further hemorrhage we do not know; that it does not always do so is certain. The bleeding for which ligature was done may continue in spite of the ligature or it may recur at a later date. The use of measures aiming at reducing vasospasm may be actually contraindicated as they could conceivably lead to renewal of hemorrhage. The risk of a permanent hemiplegia is considerably greater after ligature of the carotid artery than after ligature of the neck of the aneurysm. Finally, after carotid ligature the patient is left with just one carotid artery, which may be a potential disability. For these and other reasons we believe that a ligature of the carotid artery should not be used except in special situations where the size or location of the aneurysm makes a direct attack impossible or too hazardous.

When the patient has recovered from the initial and/or the secondary hemorrhage, which may occur while convalescing from the first attack, the question arises what to do to prevent further attacks of subarachnoidal hemorrhage. Although it is true that the danger of recurrent hemorrhage is greatest in the 2nd and 3rd week following the initial attack, the risk of another and frequently fatal hemorrhage is considerable even months and years later. In the series reported by Hyland[6] 20 per cent of the patients who were discharged alive died later from a second hemorrhage. The experiences of this clinic reported in 1952 by Norlén[9] indicate an even greater mortality rate from new bleeding. Of 22 non-operated patients with saccular aneurysms verified by autopsy or angiography from 1935 to 1948, 4 died in the hospital shortly after admission. Table 3 indicates fate of the other 18

TABLE 3

Results in cases of saccular aneurysms not operated upon

		No. of Patients
No reports		2
Dead from new bleeding (all within 3 years)		5
Dead from other disorders		1
Dead from unknown disorders		1
Alive up to 9 years after discharge		9
New attack of transient ophthalmoplegia	1	
Persistent ophthalmoplegia	1	
Disabled because of mental disorder	1	

It is also clear from the reports of Hyland and Norlén that the average life expectancy of those who are discharged alive after the first hemorrhage is short, an average of 6 years in Hyland's series and less than 3 years in Norlén's. To our mind there is no doubt that a condition that carries a mortality of about 25 per cent and a life expectancy of only a few years, and concerns mostly people in the prime of life, is in urgent need of treatment.

Since we are dealing here with a prophylactic measure, often in people who for the time being are entirely free of symptoms, any type of surgery for the purpose of preventing further hemorrhage must meet two conditions. In the first place any procedure used in such cases must be practically free of danger, that is, the mortality must be very low and the danger to function minimal. Secondly the operation should eliminate definitely the risk of further hemorrhage from the aneurysm concerned.

Ligature of the carotid artery in the neck, either the common or the internal carotid or both, meets the first of these conditions fairly well provided arteriographic studies have shown that the collateral circulation is adequate. Under this condition and in the absence of recent hemorrhage

TABLE 4

Late results of carotid ligature in 31 patients after 1–14 years

		No. of Patients
Good, full working capacity		17
Epilepsy		4
Full working capacity	2	
Mental changes	1	
Hemiparesis	1	
Mental disturbances		2
Dead from other disorders		2
Dead with signs of a new hemorrhage		6
Verified rupture of the same aneurysm	2	
Probable rupture of the same aneurysm	2	
Verified rupture of another aneurysm	1	
Cerebral hemorrhage	1	

there is considerable evidence from several clinics showing that ligature of the carotid artery carries little danger to life and function. Concerning the second condition, opinions differ. Jefferson[8] thinks that carotid ligature gives, if not complete, still a good protection against further hemorrhage since he had only 2 deaths from late hemorrhage in 142 cases. In this clinic we have not been so lucky, as is seen in Table 4, from Norlén's[9] paper of 1952. These figures indicate that probably some 10–15 per cent of the patients died later from hemorrhage in spite of carotid ligature. That some protection from future hemorrhage is afforded by carotid ligature is true or at least probable, but the degree of protection is in our opinion inadequate. It must also be borne in mind that most aneurysms on the anterior communicating artery and at the bifurcation of the middle cerebral artery are quite unsuited for carotid ligature, and these two groups form a considerable part of the lesions under discussion.

Carotid ligature still has a place in the surgery of supraclinoidal aneurysms and that is in those cases in which the aneurysm is very large and obviously unsuited for direct attack, as shown in Fig. 3.

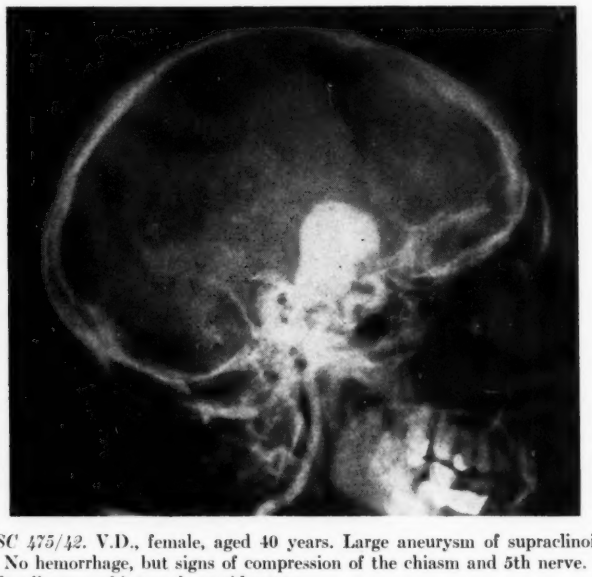

FIG. 3. *NSC 475/42.* V.D., female, aged 40 years. Large aneurysm of supraclinoidal portion of carotid artery. No hemorrhage, but signs of compression of the chiasm and 5th nerve. Disappearance of symptoms after ligature of internal carotid artery.

For about 2 years since the introduction of induced hypotension during operations we have in this clinic discarded carotid ligature except under the special conditions referred to above, and whenever possible a direct attack on the aneurysm is made. The methods employed and the results are demonstrated in Table 5.

TABLE 5

Intracranial explorations for saccular aneurysms in interval quiescent period

	No. of Patients	Method				Dead
		Lig. of Neck	Wrapping	Trapping	Explor.	
Carotid artery	15	11	1	1	2	1
Bifurcation of int. car. art.	1	1				
Proximal part of ant. cer. art.	1	1				
Ant. comm. art.	20	14	3	1	2	1
Distal part of ant. cer. art.	7	6			1	
Middle cer. art.	15	11	4			
Vertebral artery	1	1				
Multiple aneurysms with different localization	3	3				
Total	63	48	8	2	5	2

With regard to the results it should be mentioned that 1 of the 2 fatalities was caused by an extradural clot, which was diagnosed and reoperated on too late. The other patient who died came to the clinic in a semicomatose condition 2 months after hemorrhage from an aneurysm on the anterior communicating artery; death was caused by the effects of the initial hemorrhage, which had resulted in a large clot extending into the hypothalamus.

As to the procedures employed it is seen in Table 5 that ligature of the neck of the aneurysm, usually with a clip, in some cases with a linen thread, was the method most often used. To remove the aneurysm after its neck has been clipped is of course not necessary except when the aneurysm is large enough to compress important structures. This happened in 1 case in which the aneurysm had never bled and was first operated on under the diagnosis of suprasellar meningioma with bitemporal hemianopsia and near blindness. Since no arteriograms had been made, the operation had to be abandoned when an aneurysm the size of a grape was found. After arteriographic studies had established the size and exact location of the aneurysm (Fig. 4) the wound was reopened and a linen thread was tied around the neck of the aneurysm. Since it was thought that thrombosis and shrinking of the aneurysm would be too slow a process to save vision, an attempt to remove the lesion was made. When the aneurysm was opened just distal to the ligature brisk hemorrhage followed, indicating that the ligature had not been drawn tight enough. The hole in the sac was plugged with a piece of gelfoam and after a few days the wound was reopened. After removal of the gelfoam there was no more bleeding and the entire sac could be dissected out and it was removed (Fig. 5) with complete restoration of visual acuity.

Jefferson[8] has objected that a clip placed on the neck of the aneurysm in many cases actually constricts the aneurysm distal to its neck and not the neck itself. It is quite true that the clip is rarely placed flush with the wall

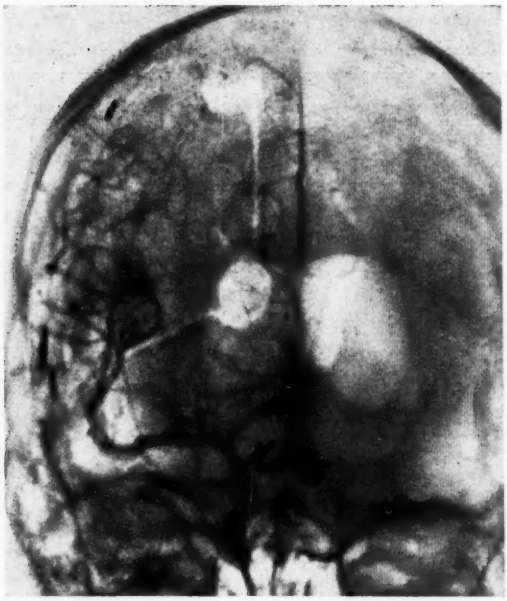

FIG. 4. *NSC 885/50.* B.P., female, aged 26 years. Aneurysm of anterior communicating artery. No hemorrhage, but compression of the chiasm and bitemporal hemianopsia.

of the parent artery but we do not believe this is of any importance because the point of rupture, when hemorrhage was recent enough to permit conclusions concerning location of the rupture, has in every case been somewhere on the top of the aneurysm and never on the neck.

Whether ligature of the neck of the aneurysm gives complete protection against future hemorrhage cannot yet be decided with absolute certainty. Control angiograms are made regularly, and if the clip has been correctly placed the aneurysm is no longer visible. It certainly must thrombose very soon and then gradually shrink, and the only point where leakage could conceivably occur would be between the clip and the wall of the parent vessel. So far we have seen no recurrent hemorrhage and consider such an event extremely unlikely.

In 3 cases it was found in the control angiogram that the clip had slipped or had been incorrectly placed. In 1 of these a second operation was performed and a new clip was placed on the neck of the aneurysm, this time with the desired result. In the other 2 cases, both aneurysms on the anterior communicating artery, nothing more was done and these cases may possibly in the future turn out to be failures.

Trapping of the aneurysm has been done in only 2 cases. When applicable it is a good method, but since it involves the loss of a piece of important

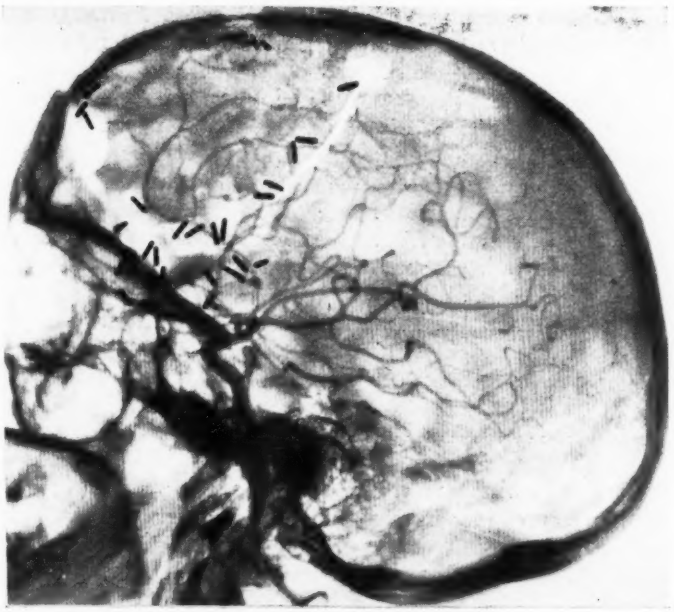

FIG. 5. *NSC 885/50.* B.P., female, aged 26 years. Same patient as in Fig. 4 after extirpation of aneurysm.

vascular channel it should be avoided when clipping the neck is possible.

Wrapping the aneurysm with muscle is an unsatisfactory method and probably does not give much protection against bleeding. It has been done only when nothing else, not even carotid ligature, was possible. With increasing experience in dissecting out the neck of the aneurysm its use has been more and more restricted.

Concerning the functional result the following complications were observed. In 2 cases of aneurysms of the anterior communicating artery, mental symptoms resembling Korsakoff's syndrome occurred after operation. In these cases the aneurysm was exposed after retracting the frontal lobe from the falx, which had necessitated the ligature of a number of veins from the frontal lobe to the sinus. In another case in which the same method of exposure was used, paralysis of the right leg and aphasia gradually set in on the 3rd day. In all 3 cases restitution of function was slow and is not yet complete several months after operation. We have therefore discarded the above-mentioned method of exposure except under special circumstances.

In some cases temporary motor or speech defects occurred or preexisting defects were aggravated, particularly in cases of aneurysms of the middle cerebral artery, but these disturbances soon cleared up and with the 3 exceptions mentioned no patient was discharged in a worse condition than

on admission. On the contrary, in cases in which hemorrhage was fairly recent and intracerebral clots of some size were evacuated, recovery of function was accelerated by the operation.

In conclusion it may be said that clipping the neck of the aneurysm or trapping meets the conditions stipulated for operations performed after the patient has recovered from the initial hemorrhage. The operation entails little danger to life or cerebral function and in all probability gives excellent protection against recurrent hemorrhage.

Concerning the technic of operation some points may be of interest. Aneurysms of the anterior communicating artery may be exposed either by elevating the frontal lobe, as in a pituitary operation, or by going in between the frontal lobe and falx. As already mentioned, this latter method has been discarded since ligature of even a few veins from the frontal lobe may induce a frontal lobe syndrome which may persist for several months. When the aneurysm cannot be exposed by elevating the frontal lobe it is better to make a small resection of the tip of the frontal pole. As a rule very little brain tissue need be removed and so far we have not observed any unfavorable reactions from this procedure. Some brain tissue surrounding the aneurysm must usually be removed by suction in order to expose completely the aneurysm and its neck.

Aneurysms of the carotid artery, its bifurcation and the middle cerebral artery are exposed by opening the Sylvian fissure after a low temporal flap has been turned. The rather thick arachnoid surrounding the carotid artery must be completely removed and the carotid artery then followed in the peripheral direction until the aneurysm is found. When the aneurysm is situated on the middle cerebral artery it is easier to proceed from the peripheral part of the Sylvian fissure.

As soon as the dura mater is being opened the blood pressure is reduced by the injection of hexamethonium bromide and if necessary the head end of the table is raised. The induction of low blood pressure should be gradual and slow and the pressure should preferably be around 40–60 when dissection of the aneurysm is begun. At this level of the blood pressure the tension of the aneurysm has been greatly reduced and there is little danger of rupture during the manipulations attending the dissection of the aneurysm and its neck. This latter structure must be absolutely clear so that it is possible to place a clip or a ligature without danger of catching adjacent

FIGS. 6 and 7. Clip holder and large size of removable silver clip.

blood vessels. Whether a clip or a ligature should be used depends upon the size of the aneurysm and its neck. We prefer a clip whenever possible and use a ligature, which is more difficult to place and to tie safely than a clip, only when the aneurysm is too large to hold a clip. Three different sizes of clips are used, depending upon the calibre of the neck of the aneurysm. It is very important that the flat silver wire used for clips has no sharp edges and that the clip holder is in perfect condition. The largest clip (Figs. 6 and 7) can be removed with the instrument used for its application and may also be used to occlude for a minute or two the parent artery centrally to the aneurysm in case this should burst during the dissection. So far this has never been necessary. A final point should also be emphasized. The clip should be applied very very gently and in closing the jaws of the clip holder the instrument should never be squeezed hard as this might easily cause the clip to cut through the very thin wall of the neck of the aneurysm.

SUMMARY

The results of operations for bleeding aneurysms of the circle of Willis depend upon the time in relation to bleeding at which operation is performed. During the first 3 weeks after hemorrhage the results of all types of operations are about the same as with conservative treatment. To the credit side of the operation must be added that if the neck of the aneurysm has been clipped the patient is protected against further hemorrhage. In case early operation is performed efforts must be made to overcome the vasospasm usually present during the first 2–3 weeks after hemorrhage.

Operations after the patient has recovered from the initial hemorrhage are to be considered as a prophylactic measure aimed at preventing further hemorrhage. Clipping of the neck of the aneurysm is the best method for this purpose. Mortality and morbidity after this operation performed 3–4 weeks after the last attack of bleeding is very low and protection against future hemorrhage is excellent.

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CASE REPORTS AND TECHNICAL NOTES

HYDROCEPHALUS IN A PREMATURE INFANT CAUSED BY PAPILLOMA OF THE CHOROID PLEXUS

WITH REPORT OF SURGICAL TREATMENT

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To warrant publication, a single case report should record a clinical experience sufficiently unusual and provocative to become valuable in itself to others who may encounter a similar problem. There is a widespread clinical impression, shared certainly by this author, that the outlook for infants born with any marked degree of cranial enlargement, as well as the outlook for infants with rapidly increasing head circumference during the first few weeks of life, is uniformly poor. Perhaps it is important, therefore, to set down an exception to this rule, and thus not only present a unique case, but in so doing, suggest an occasional happier outcome to an ordinarily discouraging and futile clinical problem.

The purpose of this report is to describe a 5-weeks premature infant, born with an enlarged head, from whom a large papilloma of the choroid plexus was removed at 4 weeks of age, or 1 week before the infant's estimated birth date, with apparent surgical cure (1 year) of a severe degree of hydrocephalus and with normal mental and physical development to date.

FIG. 1. Appearance of 5-weeks premature infant 15 days after birth. Head circumference increased almost 3 cm. during the second week of life.

C.M.C. #378515. C.T. was born 5 weeks prematurely on Sept. 25, 1951. The mother had profuse vaginal bleeding for 48 hours, at which time a diagnosis of premature separation of the placenta was made and the infant was delivered by Cesarian section at the Rutland, Vt., Hospital. The birth weight was 6 lbs., 7 oz. The baby seemed normal at birth except for a large head.

At 1 week of age it was noted that the head seemed to be growing rapidly. At that time it measured $15\frac{1}{4}$ in. (38.7 cm.) in circumference and 1 week later it had increased to $16\frac{3}{8}$ in. (41.5 cm.). At 15 days of age the infant was seen by a consultant and transferred to the neurosurgical service of the Children's Medical Center for evaluation and decision regarding the possibility or advisability of treatment.

Examination. On admission the baby was hypertonic and irritable. He weighed 6 lbs., 14 oz. The head appeared markedly enlarged with respect to the size of the trunk (Fig. 1); it measured 41.5 cm. in circumference. The anterior and posterior

fontanelles were enlarged and tense, the scalp was thin and shiny, and the veins of the scalp were congested. The cranial sutures were palpably separated and there was an extreme "cracked-pot" percussion note. The deep tendon reflexes were diffusely hyperactive. The baby's vital signs were normal and routine blood and urine analyses were not significant.

Plain roentgenograms of the skull showed an enlarged cranial vault with extremely thin bones, separation of the sutures, and soft tissue swelling in the region of the anterior fontanelle. There were no localized abnormalities or areas of intracranial calcification.

Bilateral subdural taps were negative. Ventricular puncture through the right coronal suture revealed slightly xanthochromic clear fluid at a depth of 1 cm. with a pressure of 200 mm. water. The ventricle was not located on attempted tap on the left side. Lumbar puncture revealed clear, slightly xanthochromic spinal fluid with a pressure also of 200 mm. water. The ventricular fluid protein content was 210 mg. per cent and the lumbar spinal fluid, 374 mg. per cent.

FIG. 2. Ventricular air study at 18 days of age. There is a huge multiloculated cyst in the right cerebral hemisphere, with displacement of the dilated ventricular system toward the left. Both the cyst and the ventricular system contained clear xanthochromic fluid; the color was deeper in the cyst fluid.

Ventricular air studies were performed the following day. The left ventricle was encountered at a depth of 3 cm.; air was introduced separately into this space and into the more superficial fluid-containing cavity on the right (Fig. 2). This study was interpreted as indicating a large congenital porencephalic cyst of the right cerebral hemisphere and surgical exploration was planned to attempt to establish communication between this cyst and either the lateral ventricle or the basilar cisternae.

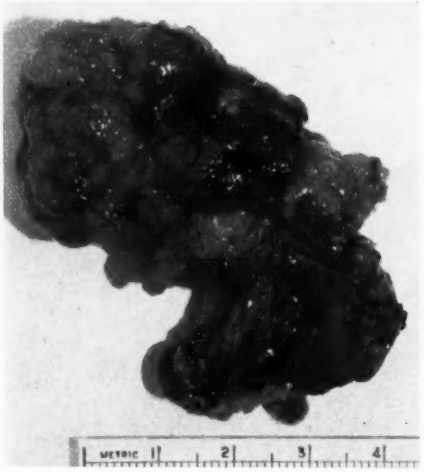

FIG. 3. Gross appearance of the tumor after excision. Actually, the size of the tumor diminished considerably as soon as its blood supply was completely secured.

1st Operation. On Oct. 16, 1951, under endotracheal ether and oxygen anesthesia, supplemented by local procaine infiltration, a right parietal osteoplastic craniotomy was performed. The infant's general condition was extremely poor, most of the operation being conducted under artificial respiration by positive pressure oxygen insufflation through the endotracheal tube. When the cortex was exposed the expected large subcortical cyst was easily identified and opened. Exploration within revealed a multiloculated cyst, the medial wall of which consisted partially of a large, irregular, reddish-purple vascular tumor and partially of a translucent membrane which when incised proved to be the lateral wall

of the right lateral ventricle. Grossly, the tumor had the appearance of a large ependymoma, though choroid plexus tumor was considered. At this stage the baby was moribund and hypothermic, and resection seemed ill-advised. Biopsies were therefore taken from two different parts of the tumor and closure was carried out. After an anxious few hours, the baby responded well and improved steadily.

Histological diagnosis of the biopsies was papilloma of the choroid plexus.

2nd Operation. Therefore, 7 days later, on Oct. 23, 1951, again under positive pressure endotracheal ether and oxygen insufflation plus local procaine infiltration, the osteoplastic flap was re-elevated. The cyst was re-entered and the tumor exposed and completely removed (Fig. 3). It was attached to the choroid plexus in the body of the right lateral ventricle and measured 4.0×3.5×2.5 cm. Since the entire lateral wall of the lateral ventricle was removed there was ample communication between the cyst and the ventricular lumen. All of the choroid plexus in this ventricle was excised. Again, after a rather uneasy first postoperative day, the infant improved steadily. His anterior fontanelle remained depressed. He was discharged home on the 10th postoperative day, at which time he weighed 8 lbs. 6 oz. and his head circumference was 40.5 cm., 1 cm. less than at the time of admission.

Pathological diagnosis on all samples of this large tumor was papilloma of the choroid plexus with no evidence of malignancy (Fig. 4).

Course. The patient has been followed for more than 1 year since operation and has remained well. There has been no evidence of increased intracranial pressure. One year after

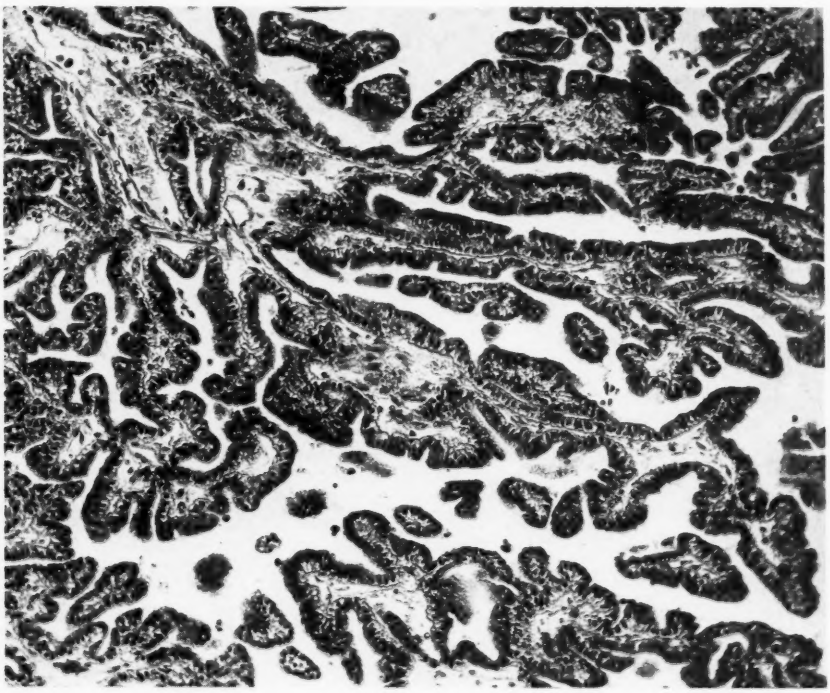

FIG. 4. Low power photomicrograph of the tumor. The histological appearance is that of normal choroid plexus, which is a core of vascularized connective tissue lined by a single layer of columnar epithelial cells. No evidence of malignancy was noted.

operation the head appears normal in size and shape (Fig. 5). There have been no convulsive seizures. Neurological examination is negative; there is no suggestion of hemiplegia or hemianopia on the left. The infant is alert and happy, sits up well, stands with help, says a few words and in general appears to be developing at a satisfactory rate.

COMMENT

That papillomas of the choroid plexus of the lateral ventricle may be accompanied by diffuse enlargement of the entire ventricular system in spite of the absence of any obstruction to the outflow of cerebrospinal fluid from the ventricles seems well established. Eight such patients have been encountered by the neurosurgical service of the Children's Medical Center, of whom the present patient is the youngest. Numerous others have been reported,[1,4–14] including at least 2 others in newborn infants.[2,3] In the latter 2 cases the diagnosis was made post mortem.

FIG. 5. Appearance of patient a little more than 1 year after excision of the choroid plexus papilloma.

If hydrocephalus in these patients is caused solely by increased rate of formation of cerebrospinal fluid in the papilloma of the choroid plexus or simply by dilatation of the ventricular system resulting from the presence of the large pulsating mass within its confines, then the hydrocephalus should be arrested promptly by successful removal of the tumor. This appears to be true in the young patient reported here, and in others in this clinic in whom satisfactory removal of the lesion has been accomplished.

It is recognized that these tumors sometimes exhibit histological features of an adenocarcinoma and may occasionally seed to other ependymal surfaces. It is possible, therefore, that the tumor in the present patient may still recur, since the follow-up period is little more than 1 year. However, resection seemed grossly complete and the microscopic appearance throughout the tumor was uniformly benign

Marked enlargement of the head at birth or rapid expansion during the first weeks of life is usually associated with gross malformation of structures within the posterior fossa, with extreme stenosis of the aqueduct of Sylvius, or with widespread failure of development of the surface subarachnoid pathways at the base of the brain. All of these conditions are unfavorable to surgical treatment in the first few weeks of life and severe hydrocephalus with destruction of cerebral substance usually ensues rapidly.

However, it is never safe to assume that a treatable lesion may not be present until certain basic tests have been performed. The presence of a markedly elevated total protein content in the cerebrospinal fluid or of xanthochromia in the absence of red blood cells should suggest the possibility of intraventricular tumor. In this clinic it is standard procedure to carry out bilateral subdural taps, combined ventricular and lumbar puncture and ventricular air studies on every infant with hydro-

cephalus who does not already exhibit gross evidence of irreversible neurological damage, no matter what the age. Routine studies of this character, supplemented by additional diagnostic and surgical pursuit of unusual clinical situations, should prevent any possibility of overlooking the occasional favorable lesion of the newborn period.

SUMMARY

A case of hydrocephalus caused by a large choroid plexus papilloma of the lateral ventricle is reported in a newborn premature infant.

Following excision of the tumor at 4 weeks of age, the infant has developed normally with no evidence of convulsive seizures, neurological deficit, or increased intracranial pressure for 1 year to date.

This patient appears to represent an exception to the extremely poor prognosis ordinarily entertained for newborn infants with rapidly advancing cranial enlargement.

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FAMILIAL HEMANGIOBLASTOMA OF THE CEREBELLUM

PEDIGREE OF TWO FAMILIES

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The hereditary basis for the occurrence of some hemangioblastomas of the central nervous system has been well established. However, the reports of such families are sufficiently scarce to warrant recording two additional families having members of several generations afflicted with this lesion.

Case 1. M.V., a 22-year-old married woman of Hawaiian extraction, was first admitted to the San Francisco Hospital on Aug. 1, 1948, complaining of occipital and frontal headaches accompanied by nausea and vomiting for the previous 3 months. Physical and neurological examinations were negative. The family history obtained from the patient indicated only that her father had died of a brain tumor of unknown type. Subsequently, it was revealed that several members of the paternal branch of the family in the preceding two generations had had brain tumors (Fig. 1).

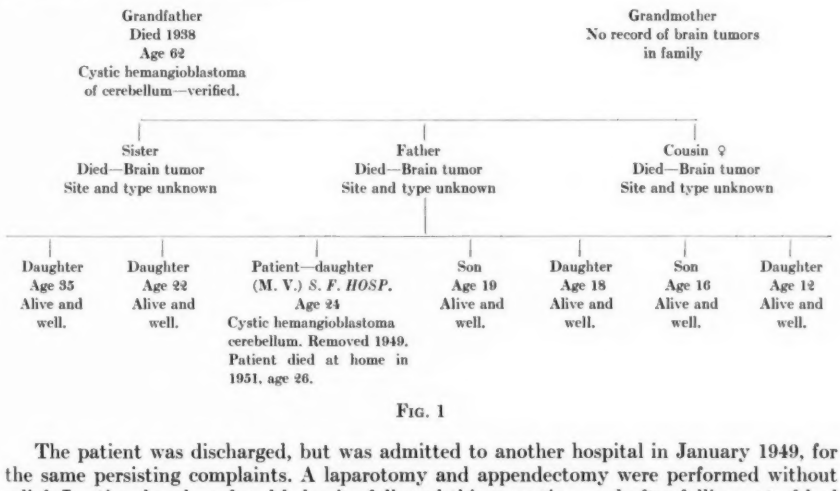

FIG. 1

The patient was discharged, but was admitted to another hospital in January 1949, for the same persisting complaints. A laparotomy and appendectomy were performed without relief. Irrational and confused behavior followed this operation, and after falling out of bed on her 6th postoperative day, she was readmitted to the San Francisco Hospital.

Examination. At this time, papilledema was noted, accompanied by bilateral extensor plantar responses and bilateral external rectus palsies. Roentgenograms of the skull were normal. Lumbar puncture demonstrated a CSF pressure of 290 mm. of water, xanthochromic fluid, 235 RBC/c.mm., and a protein content of 450 mg. per cent. Because of the fall, a diagnosis of subdural hematoma was entertained but trephination was negative. Subsequent ventriculography revealed a uniformly dilated ventricular system without deformity although the 4th ventricle was poorly filled with air.

Course. Following ventriculography the patient improved. The headaches and diplopia disappeared and the papilledema became less marked.

She was next seen 10 months later, again complaining of headaches and difficulty with speech. Papilledema was marked (6 D. bilaterally), and examination disclosed truncal ataxia and palsies of the 5th, 6th, 7th, 9th, 10th, and 12th cranial nerves on the left.

Operation. An exploration of the posterior fossa was performed on Oct. 27, 1949, and a large cystic hemangioblastoma of the left cerebellar hemisphere was removed. A similar noncystic tumor involved the upper cervical cord and lower brain stem. A biopsy was made of this.

Course. Roentgen therapy was given following a stormy postoperative course. She remained well until August 1951, when she suddenly expired at home. Permission for an autopsy was not granted.

In the following two cases the patients were brother and sister and were both hospitalized at the University of California Hospital at the same time with almost identical histories and tumors.

Case 2. E.M., aged 40, brother of Case 3, was admitted in April 1950. He had suffered from increasingly severe occipital headaches for the previous 4 months. He had noted staggering gait for 2 months.

Examination. Positive neurological findings were 2 D. of papilledema bilaterally, slight truncal ataxia, and fine nystagmus on right lateral gaze. Ventriculography demonstrated a symmetrical hydrocephalus and obstruction of the 4th ventricle.

1st Operation. An immediate suboccipital craniectomy was performed, and a large midline cyst of the cerebellar vermis with a nubbin of tumor at its superior pole was encountered and removed.

Microscopic examination of the tumor disclosed the typical picture of hemangioblastoma.

Course. He remained well for 2½ years, when he reentered the hospital with signs of recurrent tumor.

2nd Operation. Re-exploration disclosed a large solid tumor involving the left cerebellar hemisphere. Successful removal was accomplished and postoperative irradiation was instituted.

Course. At present, the patient is well.

Case 3. V.N., aged 35, sister of Case 2, was admitted 2 weeks after her brother's first admission. Her complaints were similar to his, namely, occipital headaches, staggering gait, morning vomiting, and vertigo.

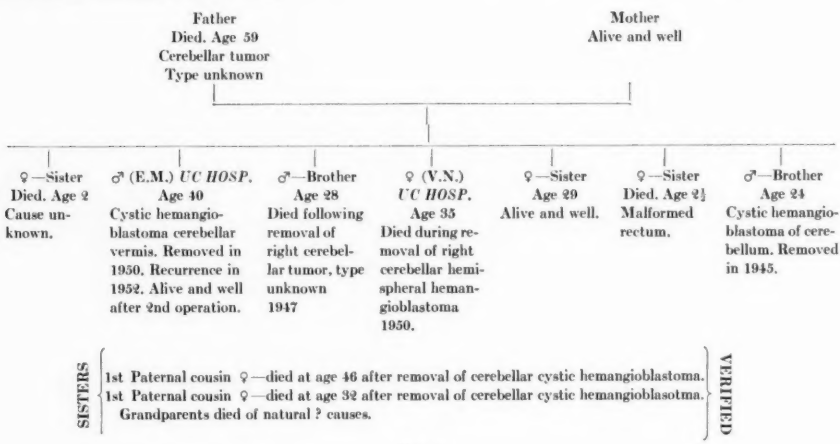

FIG. 2

Examination. Two D. of papilledema were present bilaterally and moderate truncal ataxia was noted. Ventriculography demonstrated a block in the posterior fossa.

Operation. Immediate suboccipital craniectomy disclosed a cystic and solid tumor involving both the cerebellar vermis and the right cerebellar hemisphere. The patient was operated upon in the sitting position and unfortunately expired during the procedure.

Autopsy disclosed air embolism as the cause of death. The cerebellar tumor was a cystic hemangioblastoma. In addition, there were present cystic adenomata of the pancreas.

The family pedigree of Cases 2 and 3 is shown in Fig. 2. In none of the 3 cases was any angiomatous involvement of the retina demonstrated. It is of interest that in each of these families, symptoms referable to the tumor appeared at a younger age in successive generations. This tendency has been commented upon in previous reports of such families.[1]

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BRAIN NECROSIS FOLLOWING X-RAY THERAPY

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Brain and nerve tissue are usually resistant to direct damage by x-ray radiations, even with high dosages.[1,4,11] There is increasing evidence, however, that in rare instances the vascular system of the brain can be severely damaged by irradiation.[2,7,8,10,12,14,15] We have recently studied a case that we feel demonstrates this phenomenon. The patient, originally treated for a basal cell epithelioma of the scalp with x-ray therapy, subsequently showed evidence of an underlying, expanding, intracranial lesion. At operation a non-tumorous mass was removed from the temporal lobe; microscopic study of this mass revealed startling blood-vessel changes.

CASE REPORT

Past History. L.C., a 45-year-old white female, was first seen in November, 1945, when a biopsy diagnosis of a basal cell epithelioma of the right temporal scalp was made. Superficial x-ray therapy (2500 r) was given to the lesion on Nov. 12, 1945. During the next 2 years, similar lesions appeared over the neck, shoulder, chest, and abdomen. These were treated as they appeared, either by local excision or by electrofulguration. The tumor in the right temporal area reappeared in 1948. Further x-ray therapy was felt unwise because of the marked scarring, atrophy, and depilation of the skin in that area. The lesion was excised and again a diagnosis of basal cell epithelioma was made. She had no complaints suggesting intracranial disease at that time.

Present Illness. On July 30, 1948, the patient was brought to the hospital because of lethargy, confusion, incontinence, and headache of 4 weeks' duration. The onset had been marked by disabling pain and weakness in both legs and was followed by urinary and fecal incontinence. Daily convulsive seizures, lasting about 10 minutes, started 2 weeks before admission; there were no recognized aura or localizing manifestations.

Examination. She was a well-developed, obese, white woman with marked facial hirsutism. Her pulse was 80, respiratory rate 18, and B.P. 145/85. She was apathetic, lethargic, and

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confused. Her speech was monosyllabic and explosive. There was a restless, continuous, picking motion of her hands. Slight, bilateral exophthalmos was present and the left pupil was larger than the right. Both pupils reacted sluggishly to light. There was early bilateral papilledema. Cooperation was too poor to test visual acuity or visual fields. There was left facial flattening and a minimal, but definite, weakness and hypesthesia of the left arm and leg with decreased tone and increased tendon reflexes on that side. She was right-handed. There was a dorsal Babinski response bilaterally. The patient was unable to care for herself and was incontinent of urine and feces. In the right temporal area of the scalp was a depilated area, 5×5 cm., of thin, atrophic, scarred skin with a central ulcer 1 cm. in diameter.

Urine was normal, Hb. was 13.5 gm. per cent, and WBC count was 7,100 with 66 per cent segmented neutrophiles. The blood Mazzini and spinal fluid Kolmer tests gave negative findings. Lumbar puncture showed an initial pressure of 240 mm. of CSF, which had a total protein of 160 mg. per cent. The spinal fluid gold curve was 1112210000. Roentgenograms of the skull showed a midline calcified falx cerebri and no apparent pineal shift. Ventriculography on Aug. 10, 1948 showed no filling of the right ventricle, minimal dilatation of the left ventricle, and a shift of the entire system, including the 3rd ventricle, to the left. The posterior 3rd ventricle was not visualized. The total protein of the left ventricular fluid was 25 mg. per cent.

Course. During the next 5 days, the patient's status became steadily worse with an increasing left hemiparesis and slowing pulse and respirations.

1st Operation, Aug. 16, 1948. Because of the suggestion of a 3rd ventricle or right posterior parietal lesion, an exploratory craniotomy was done through a right posterior parietal bone flap. The most anterolateral portion of the cortex exposed was slightly yellow. The rest of the exploration, including examination of the 3rd ventricle through the corpus callosum, was

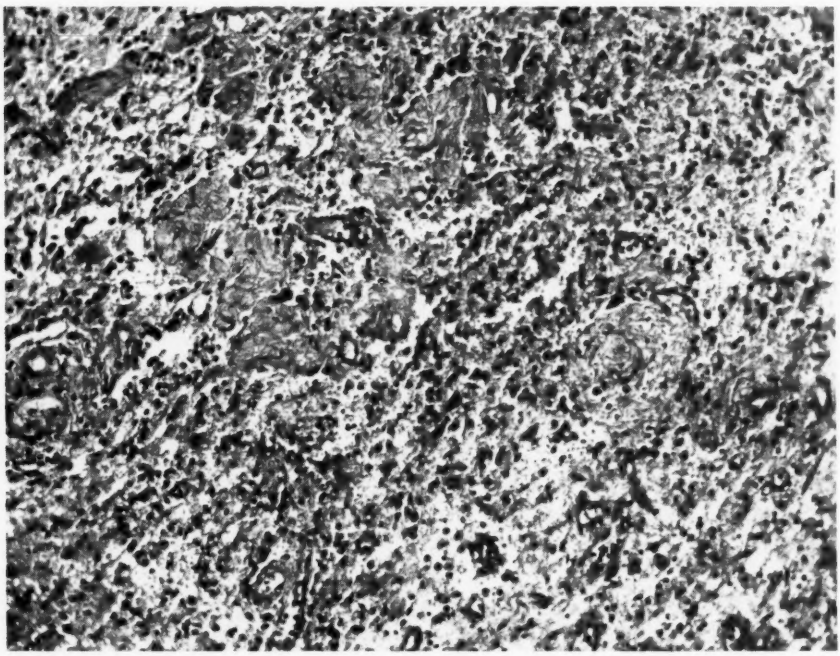

FIG. 1. Extreme thickening and hyalinization of small blood vessels. Infarction of brain with exudate containing many gitter cells. Changes suggesting amyloid degeneration. (van Gieson×215)

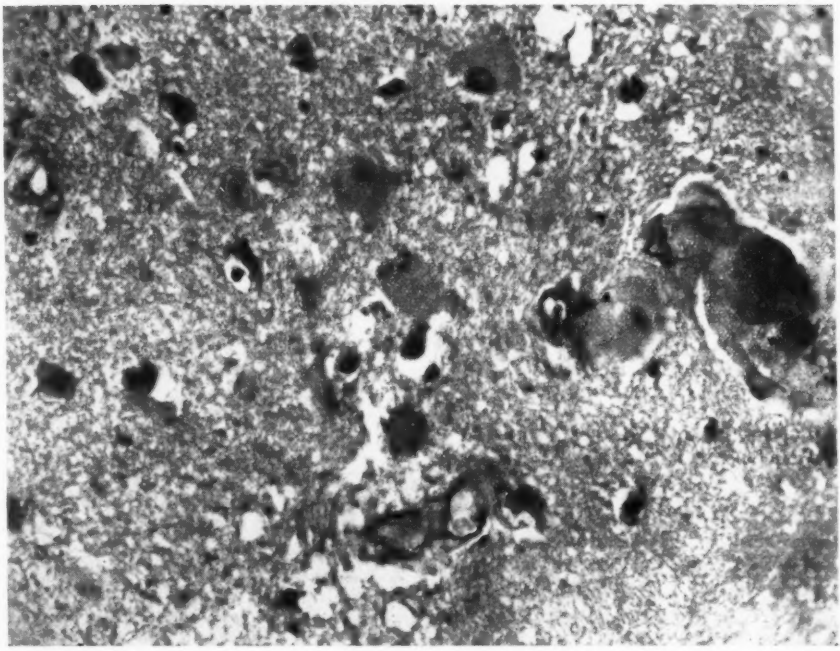

FIG. 2. Edematous brain adjacent to area of necrosis. Note swollen astrocytes and separation of elements of brain tissue. Normal blood vessels. (Bodein ×185)

negative. The intracranial pressure was critically high and required removal of the bone flap as a life-saving procedure.

Postoperative Course. The signs of increased intracranial pressure subsided rapidly, and there was no further progression of her neurological findings.

2nd Operation, Sept. 13, 1948. A right temporal craniotomy was done. A circular area of the right parietotemporal brain cortex directly underlying the irradiated skin was grossly abnormal. This area was approximately 4 cm. in diameter. It was avascular and yellow-gray. It both looked and felt hard. The borders were fairly clear, but there was no capsule. The abnormal tissue was resected to a depth of 4 cm. Immediate frozen section of this tissue showed no evidence of tumor, but, rather, brain necrosis and marked blood-vessel changes. Bleeding was minimal. The tissue in the bed of the resection appeared to be nearly normal. There was no gross evidence of infection or cyst formation. Needle biopsies of the surrounding brain were taken. Evidence of increased intracranial pressure was totally absent at this operation.

Course. Her postoperative course was stormy, but showed gradual improvement. The left hemiplegia, left hemihypesthesia, and incontinences remained unchanged. However, her lethargy, seizures, confusion, and speech impairment all disappeared.

At a check-up examination on April 18, 1953, there was no evidence of progression of her disease. The decompression was soft and pulsating. The CSF pressure was 80 mm. of spinal fluid. There was slight improvement in the left hemiplegia.

Pathological Findings—Gross. The surgical specimen removed during craniotomy on Sept. 13, 1948 was composed of two pieces of firm, yellow-gray tissue. The larger measured 3×3 ×2.5 cm. and weighed 11 gm. The smaller measured 1×1.5×0.4 cm. and weighed an esti-

mated 2 gm. The surface made by cutting was yellow-gray, firm, and avascular with soft, gray-pink tissue at the margins.

Microscopic. The meninges were thickened by fibrous tissue in which there were many lymphocytes, macrophages, and a few scattered neutrophilic leukocytes. The characteristic feature of the underlying brain tissue was the marked replacement of the normal structures by a homogeneous amorphous substance. Silver stains demonstrated a striking decrease in nerve and glial fibers, those remaining being in the subpial area and around blood vessels. In smaller areas of brain tissue there was cellular necrosis with masses of gitter cells and swollen astrocytes (Fig. 1). Iron stains revealed a few iron-containing granules in macrophages adjacent to blood vessels in the necrotic brain. Immediately adjacent to the necrotic areas the cortical tissue was edematous, showing swollen astrocytes and separation of nerve fibers (Fig. 2).

The blood vessels of the meninges and of the underlying brain showed varying degrees of thickening and luminal narrowing (Fig. 3). The thickening involved any or all layers. With hematoxylin and eosin stain some vessel walls appeared homogeneous and pink, suggesting amyloid degeneration, a fact further suggested by special stains. Weigert stains for elastin

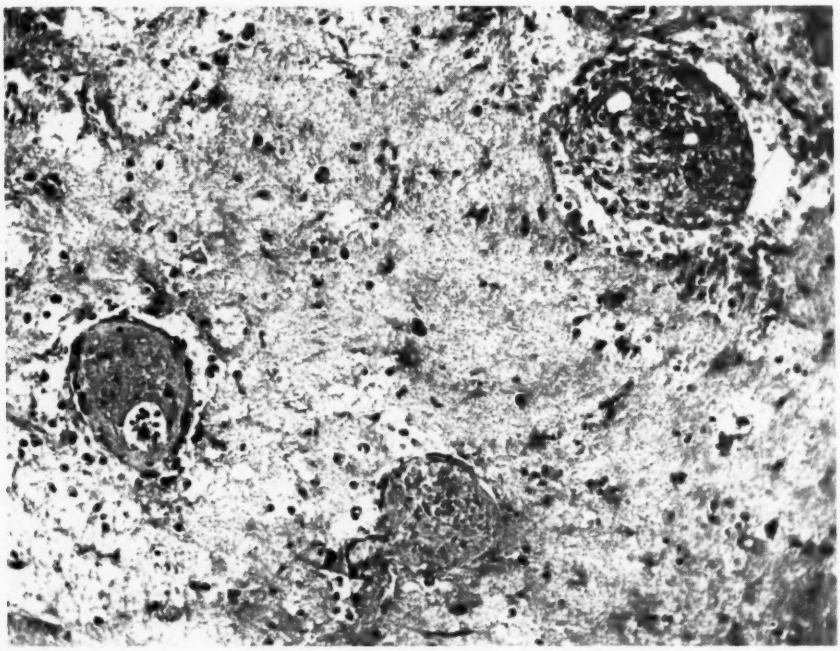

FIG. 3. Section of three arteries with thickened walls and narrowed lumina. (van Gieson ×215)

demonstrated fragmentation and separation of elastic fibers, as well as complete absence of elastic fibers in some vessels. Muscle fibers were replaced to a varying degree by fibrous or hyalinized tissue. Many of the vessels, particularly those deep in the cerebrum, were composed of thick, homogeneous hyaline material with absent to extremely narrowed lumina (Fig. 1). Thickening of the adventitia was prominent in some vessels (Fig. 3). An exudate of lymphocytes was present in some of the thick-walled vessels and in the adjacent connective tissue (Fig. 3). There were striking examples of endothelial swelling. Groups of telangiectatic

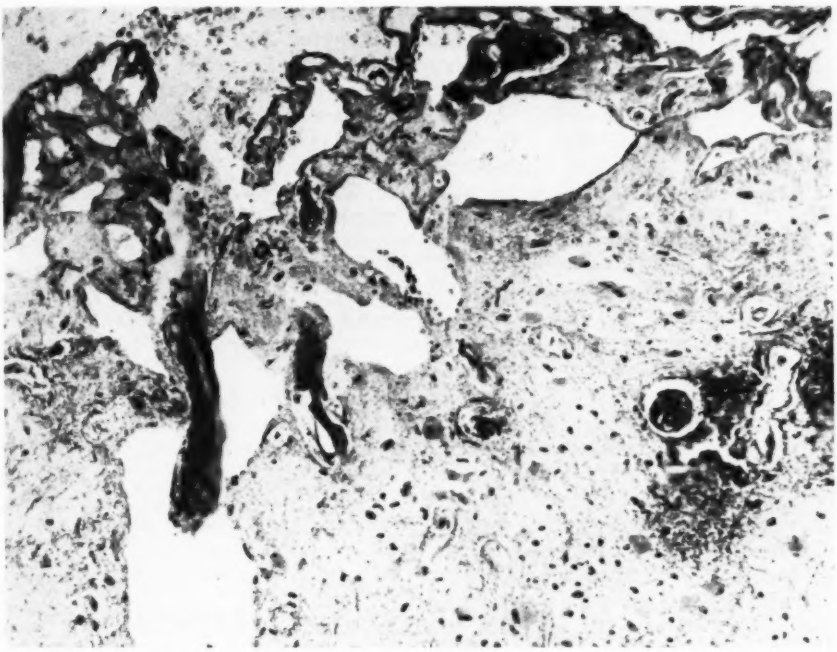

FIG. 4. Telangiectasia of subpial blood vessels. Necrosis of adjacent brain. (Bodein ×240)

vascular spaces were present in the subpial region (Fig. 4). Vessels in the edematous brain bordering on the necrotic tissue were normal (Fig. 2).

IRRADIATION DATA

This patient received superficial x-ray therapy $2\frac{1}{2}$ years prior to the development of signs of intracranial disease. She received only one treatment. The details of this x-ray treatment, given on Nov. 12, 1945, are:

Size of field (right temple)	5×5cm. (approximate)
Target skin distance	25 cm.
Filter	Inherent only (1 mm. aluminum equivalent)
Kilovolt	120
Milliamperes	20
Time	15 minutes, 30 seconds
Air dose, at skin	2500 roentgens

The skin dose of irradiation was 2925 roentgens. At 2 cm. (approximately at the surface of the temporal lobe), the depth dose was 1287 roentgens. At 5 cm. (approximately the maximum depth of operation), the depth dose was 526.5 roentgens.

DISCUSSION

The pathological studies in this case strongly suggest that the extreme blood-

vessel changes were the cause of the cerebral necrosis. Such vascular changes have been recognized as the probable basic factor in certain instances of radiation brain damage, both experimentally[2,7,8,12,18] and clinically.[9,13–15,17] It must be pointed out that many reports of radiation brain necrosis describe no vascular damage,[1,5,16] and other reports place little or no emphasis on minimal vascular changes observed.[10,15,17,18]

The main characteristics of the blood-vessel changes were: (1) hyalinization and thickening of the walls with resulting luminal narrowing in all stages of development, including actual occlusion of the vessel; (2) suggestive amyloid degeneration of the vessel walls; and (3) telangiectasis of subpial vessels. Such progressive hyalinization and thickening of vessel walls is recognized as characteristic of irradiation cerebral vascular damage.[9,13–15,17] Amyloid degeneration in vessel walls as an irradiation effect is recognized, though not widely reported.[3,6] The telangiectasia, found here in the subpial region, is seldom reported as part of radiation brain damage. It is significant that the blood vessels were normal in the brain immediately adjoining the necrotic lesion.

The brain necrosis was a progressive ischemic infarction, apparently the result of vascular insufficiency and even successively occurring vascular occlusions. The recently infarcted areas, where gitter cells were massed, contrasted sharply with the older areas of infarction where no cellular activity existed.

The time interval between irradiation and the onset of symptoms was $2\frac{1}{2}$ years. Others have observed a similar interval.[14,15] With a progressing, obliterative, vascular disease such a time interval seems reasonable since the process was obviously in progress long before the onset of symptoms. Delayed manifestation of right temporal lobe disease is not unusual.

We believe the acute, progressive, clinical manifestations of this lesion were caused by two major factors: (1) The ischemic necrosis of the right temporal lobe had become extensive enough to be clinically evident. (2) Cerebral edema of unexplained origin, possibly related to vascular occlusion, caused the signs of increased intracranial pressure and ventricular shift. We feel the cerebral edema was comparable to the edema that often accompanies brain tumors, but that the relatively benign postoperative course of 5 years mitigates somewhat against a "missed" brain tumor.

The findings we have described are consistent with the pathologic diagnosis of brain necrosis resulting from excessive radiation. The incidence of such a complication secondary to x-ray therapy is exceedingly rare.

We must assume that for this particular patient, this particular dosage as applied was too great for toleration by the cerebral vascular system. We do not feel that the circumstances warrant the simple interpretation of overdosage. Rather, we feel that certain obscure factors were involved. Whether these factors concern abnormalities of the specific cerebrovascular system under treatment, must at this writing remain a matter of conjecture.

SUMMARY

1. A case report has been presented in which evidence of a progressing intracranial lesion developed $2\frac{1}{2}$ years following superficial x-ray therapy of 2500 roentgens for a basal cell epithelioma of the right temporal scalp. Operation confirmed a right temporal lobe brain lesion immediately underlying the irradiation scar of the scalp.

2. The pathological feature of this lesion was ischemic infarction of the brain caused by progressive, obliterative, vascular disease. These changes were sharply limited to the brain immediately beneath the irradiated scalp.

3. Significant features of this case have been discussed and correlated briefly with pertinent reports on radiation brain damage.

4. No basic cause for the radiation vascular damage has been established. It has been suggested that the vascular changes may have been the result of individual causes more subtle than the relatively simple conclusion of overdosage.

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OSSIFYING SUBDURAL HEMATOMA

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Calcification within a chronic subdural hematoma occurs rarely.[1,3,8,10] Although Rokitansky[13] wrote over a century ago. . . . "Die Wände des Sackes, zumal die an der harten Hirnhaut haftende Wand, werden zuweilen der Sitz von Verknöcherung, d.i. von Knochen-Concretion in der Form von Platten," only a few similar examples have been noted. In a review of the literature we have encountered 3 reports of histologically verified ossification within a chronic subdural hematoma[2,6,15] to which we wish to add a fourth case.

CASE REPORT

A 23-year-old white man was involved in a motor car accident on Mar. 29, 1942. He was dazed for a moment and about an hour later experienced left frontotemporal headache. He went to his work in a steel mill the next morning but was unable to continue after 30 minutes because he could not stand the noise. Roentgenograms of the skull were said to have disclosed a "fracture." He remained in bed for 2 weeks and 6 weeks later was able to return to his work, at which time his headache had disappeared. He was inducted into military service on Aug. 15, 1942, and went through training without difficulty except for bothersome headaches on the mornings following long study for examinations. While on duty as an armorer-gunner on a bomber in North Africa in August 1943, he struck his head against a radio desk during a difficult landing, was dazed for a few minutes, and was not aware of any other immediate complaints. In October or November 1943, he noted feelings of "tightening up" and a sense of pressure in the head which was attributed to "operational fatigue" and which was sometimes associated with a sensation of ringing in the head and some blurring of vision. When he had completed 64 missions he was returned to the United States where he mentioned that he had headaches; X-rays of the skull were taken and he was referred for physical evaluation in October 1944. Neurological findings were not remarkable, although both plantar responses tended to be extensor at times.

X-ray Examination, Oct. 10, 1944. Roentgenograms of the skull showed a somewhat mottled plate of calcification beneath the inner table on the right, extending from the anterior portion of the frontal bone posteriorly beneath the parietal bone for a distance of 13 or 14 cm. The pineal body was very faintly calcified and could be visualized only on the lateral view. The pituitary fossa was normal in size and shape and there was no erosion of the clinoids or of the petrous pyramids.

Psychometric Examinations, Oct. 18, 1944. Results of the tests pointed to an individual of average intelligence. Notable inconsistencies in performance indicated a very marked impairment of recent memory ability and some degree of weakness in the sphere of social judgment and comprehension. Performance of memory tests corroborated these findings in indicating average remote memory ability, but striking and consistent impairment of recent memory functioning. The cooperation of the patient was adequate and results were considered representative, but positive findings suggested that the results did not reflect the optimum level for the patient at the time.

Wechsler-Bellevue Verbal Scale, Form B	Mental age: Average adult. IQ 105
Wells Memory Test	Remote Memory MQ: 97
	Recent Memory MQ: 67
Cornell Selectee Index	Score 30/15
Aphasia Test Battery	Negative

Pneumoencephalogram, Oct. 23, 1944. The entire ventricular system was filled and there was also some air in the subdural space. The lateral ventricles were displaced slightly toward the left and the upper surface of the right lateral ventricle was slightly depressed. The left lateral ventricle appeared to be slightly dilated as compared to the right. Some of the subarachnoid pathways appeared to be obliterated on the right side and there was dilatation of most of the pathways on the left.

The CSF was under normal pressure; cells, total protein, colloidal gold reaction and serology were unremarkable.

EEG, Dec. 12, 1944. The EEG was normal. The dominant wave frequency was 8–10 cycles per sec. with maximum amplitude of 30 to 50 microvolts. There was no evidence of focal or diffuse abnormalities. There was no change in pattern on overventilation.

Operation, Dec. 19, 1944. Under local anesthesia a large bone flap, centered in the right temporal region, was made. On elevating the flap a somewhat bluish discoloration of the dura mater was disclosed, extending for a distance of 5 cm. from the midline. This area was stony hard to touch, although at the limits of its extent the dura mater was soft and compressible. A dural flap was outlined just beyond the inferior limits of the lesion and when it was reflected it was found to consist of what appeared to be two layers, although the inferior layer was in all probability the outer capsule of the lesion. The dura mater was carefully dissected back and reflected up to the longitudinal sinus (Fig. 1). The calcified mass extended up to the longitudinal sinus and at its attachment it was perforated by the large veins from the Rolandic area where they entered the sinus. The calcified mass was not exposed in its complete extent, a small portion still remaining under the bone at the anterior and posterior limits of the flap.

FIG. 1. Appearance of operative field showing ossifying subdural hematoma with dura mater reflected upward along the longitudinal sinus.

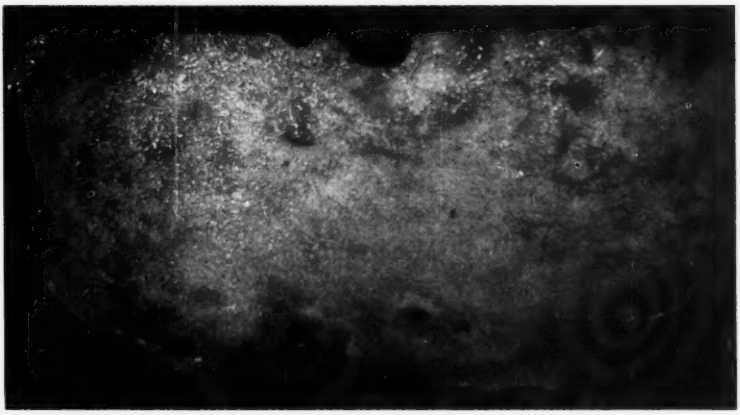

FIG. 2. Photograph of specimen removed at operation.

The main part of the mass was freed from the remaining ends with rongeurs. It was reflected toward the midline, and after bits of bone were nibbled from around the large veins entering the longitudinal sinus, it was removed by careful dissecton.

The patient made an uncomplicated recovery.

Pathological Examination. The specimen consisted of an irregular ragged piece of hard material grossly resembling bone, and it was curved corresponding to the contour of the skull (Fig. 2). The overall measurements were 1½×7 cm. and 3–4 mm. in its average thickness. The convex surface was somewhat granular in appearance, almost velvety. There were two fenestrations measuring 5 mm. in their greatest diameter. The concave side was mottled and very irregular, and several of these mottled portions were pearly white. Surrounding the firm middle portion, there were several irregular edges that seemed to be composed of hard fibrous tissue.

On microscopic examination the specimen was found to be composed of masses of hyaline fibrous tissue undergoing calcification and osseous transformation. There were some rather large spicules of bone.

A roentgenogram of the specimen is shown in Fig. 3.

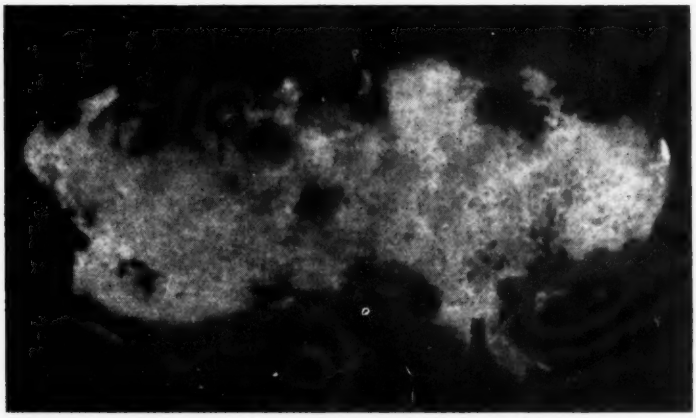

FIG. 3. Roentgenogram of specimen removed at operation.

DISCUSSION

Goldhahn[6] excised from an 11-year-old child an intracranial mass with a bony shell, the presumed end-product of a "hemorrhagic pachymeningitis interna." Histological examination in Boyd and Merrell's[2] case revealed heavy deposits of calcium in the thick hyalinized connective-tissue wall, some of which had undergone ossification. Typical true bone was found by Wertheimer and Dechaume[15] in portions of the walls of a calcified subdural hematoma removed by operation.

In some reports dealing with the presence of calcification in the walls of chronic subdural hematomas a statement regarding a histological examination is absent. Schüller's[14] article contained photographs of two preparations from the Vienna Pathological-Anatomical Museum, demonstrating in one a "bony shell" around an organized subdural hematoma and in the other a "bony plate" over a cerebral convexity. Although Dyke and Davidoff[5] described the roentgenographic appearance of "a mass of bone 1 to 2 cm. thick adjacent to the inner table of the skull," which they felt represented an example of a chronic or ossified hematoma, this patient apparently was not operated upon and the lesion was not verified. In Critchley and Meadows'[4] case the nature of the "calcified plates" encountered at operation was not fully defined. The appearance of the gross specimen in the report of Mosberg and Smith[9] suggests the possibility of ossification, but their report stated only "microscopic examination confirmed the diagnosis of calcified subdural hematoma."

A tendency to use the terms *calcified* and *ossified* interchangeably has been noted.[5,12] The term ossified or ossifying should be reserved for those hematomas that show evidence of bone formation on microscopic examination, since calcification without ossification may also occur.[7,11] Ossification when present may be considered a terminal phase in the organization of a chronic subdural hematoma and is usually preceded or accompanied by hyalinization and calcification, especially of the peripheral portions. It has been assumed that hematomas of long duration are most prone to calcify and ossify,[5] although the data from some cases are obscure.

In this case, no obvious history of a likely causative head injury prior to 2½ years before operation was elicited.

SUMMARY

The clinical history of a 23-year-old man who had an ossifying subdural hematoma has been presented. The literature pertaining to ossification and calcification within chronic subdural hematomas has been reviewed and the desirability of histological study in such cases emphasized.

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REMOVAL OF BULLET FROM THE BRAIN BY GRAVITY

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(Received for publication January 13, 1953)

In an excellent historical review of the literature and a report of personal experiences, Wood[6] emphasized that spontaneous movement of metallic foreign bodies in the brain is strongly suggestive of abscess formation. Other authors[1–5] have discussed the use of gravity in attempting to bring about migration of metal objects to surgically more accessible regions of the brain. Varying degrees of success have been reported. However, we have been unable to find a report of successful movement of a bullet by gravity alone from a point within the brain to a position beneath the scalp. This is a report of the movement of a bullet from a location 8 cm. within the brain to a position beneath the scalp where it was easily felt and removed. This occurred without the formation of abscess or hematoma.

CASE REPORT

B.M., a Latin American male aged 7, was admitted to Parkland Hospital on July 28, 1952, within 30 minutes after having been wounded by a pistol bullet.

Examination disclosed no neurological abnormalities. Radiographs (Fig. 1) showed the bullet to have entered the parietal region of the skull approximately 2½ cm. anterior to the lambda and 2½ cm. to the left of the mid line. A few small fragments of metal were left in the surface of the brain near the skull wound but the major portion of the bullet ranged downward, forward and slightly laterally, coming to rest in the left temporal lobe of the brain 8 cm. from the point of entry through the skull.

The wound was debrided with removal of small metal and bone fragments from the brain but no attempt was made to remove the major fragment of bullet in the temporal lobe. The dura mater was left open but the galea was closed tightly with silk sutures.

[Dr. H. F. Brewster, who was chief of surgery at Navy 128, Pearl Harbor, T.H., in January 1944, told one of us (L.B.B.) that it was possible to remove bullets by gravity, and at that hospital and at that time there were 2 cases in which bullets were removed by gravity.]

During the next 12 days, the patient was kept on his back as much of the time as was possible without using restraint, and the foot of the bed was elevated. This was done to place the bullet nearly vertically above the point of entry. At the end of 12 days, the bullet could

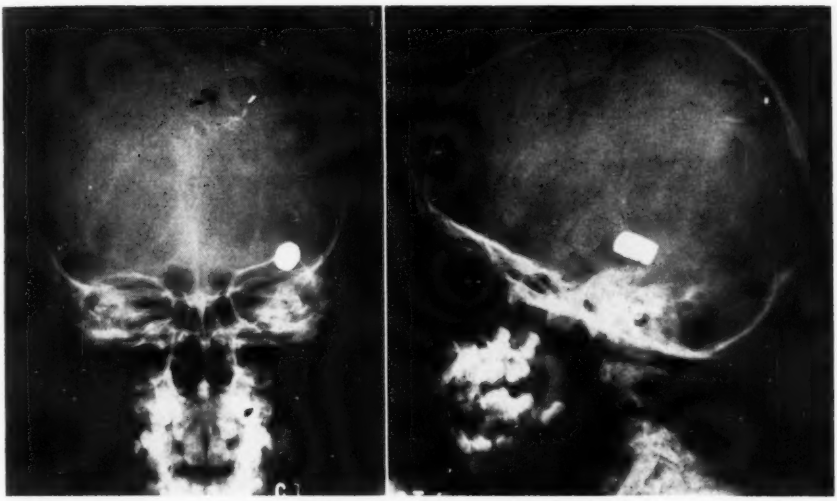

FIG. 1. Radiographs showing the bullet in the left temporal lobe of the brain. Wound of entry is indicated by arrow.

be felt beneath the scalp and radiographs made 14 days afterward (Fig. 2) showed the bullet to lie outside the skull beneath the scalp. It was easily removed and there was no evidence of abscess or hematoma formation.

The patient was discharged the following day and was last seen in Clinic on Nov. 19, 1952, at which time he appeared normal.

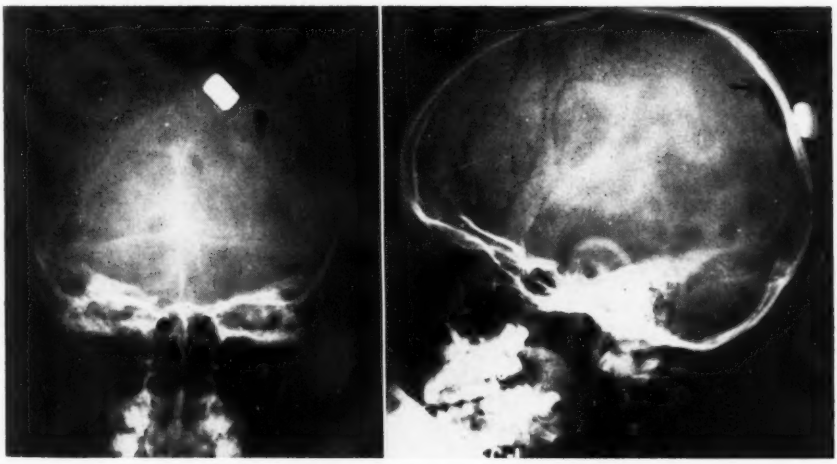

FIG. 2. Radiographs showing the bullet beneath the scalp outside the skull. Wound of entry is indicated by arrow.

SUMMARY

A review of the literature reveals various theories that have been suggested to explain the movement of bullets in the brain. Abscess and hematoma formation, location inside a ventricle and brain softening have been suggested as causes for movement. Wood[6] stressed the importance of abscess formation occurring in association with movement of metallic foreign bodies in the brain.

This is a report of a case in which gravity was used intentionally and successfully in moving a bullet from its location 8 cm. within the skull backward along the path of entry to a point beneath the scalp from where it was easily removed, and in which there was no abscess or hematoma.

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CERVICAL TRAPEZE

AN APPARATUS FOR AMBULATORY TREATMENT OF FRACTURES OF THE CERVICAL SPINE

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(Received for publication October 20, 1952)

The ambulatory treatment of the patient with a fractured neck who has no neurological injury, or whose neurological injury is not so severe but what the patient can be given such treatment, is an infrequent but desirable type of therapy. Having treated a goodly number of these patients with various types of traction devices that permit ambulation, it occurred to us that it would be well to develop a frame that could be used on patients of different builds. We have applied such traction 48 hours after the injury. The apparatus is a simple, adjustable, lightweight frame which is quite easy to employ and which will maintain adequate traction on the head so that the patient can be in any position.

The frame (Fig. 1) consists of vertical (A) and horizontal (B) tubes which are adjustable for anatomical variations in length of neck and size of head. Malleable straps (C) are attached to the base of the frame to be incorporated in a plaster jacket or full body cast. Attached onto the frame is a pulley device (D) with a flexible cable (E) and a small spring-scale (F) so that traction can be maintained at any desired weight. A turnbuckle (G) is interposed between the cable and the spring-scale to allow a finer weight-traction adjustment.

* 350 East Broad Street, Columbus 15, Ohio.
† Wilmington, Ohio.

The head frame* may be applied with the patient in a horizontal or vertical position, depending upon the particular condition of the patient and the equipment available to the surgeon. In either case the plaster or plastic cast is applied while the head remains in traction. After application of the major portion of the plaster body jacket, the malleable metal straps (they are actually malleable ribbon retractors) are incorporated into the jacket over the shoulder. As soon as the cast is dry enough to stand pressure the tubular frame is assembled and fastened to the metal straps in the cast. The cable is attached to the head tongs by means of a hook (H) and the desired amount of traction is applied, tightening the cable at a clamp (I) connecting the cable to the spring-scales, the latter being connected to one side of the frame (J). A finer adjustment can be obtained by a few turns on the turnbuckle placed between the cable and the scales. The patient is allowed to sit, stand or walk whenever his condition permits (Fig. 2).

* The cervical trapeze is being manufactured by the Zimmer Manufacturing Company of Warsaw, Indiana.

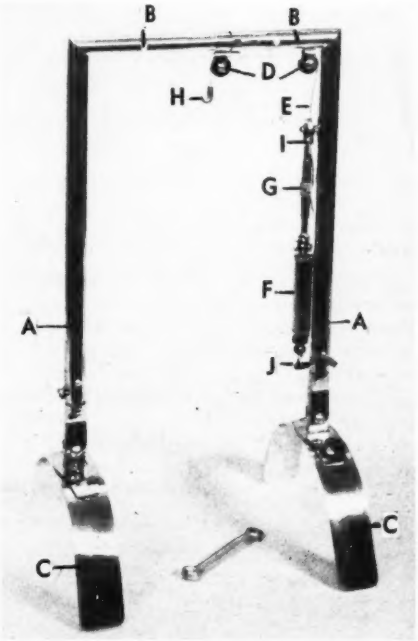

FIG. 1. Cervical trapeze (described in text).

FIG. 2. Cervical trapeze applied with body cast to patient with fracture dislocation over C4–5 without neurological complications.

A RUBBER VENTRICULAR CANNULA AND INDWELLING CATHETER

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(Received for publication December 11, 1952)

The cannula herein described was devised to tap a ventricle and also to be left in situ for injection or removal of air or fluid.

The cannula* is made rigid by a metal stilette.† The end of the soft but not collapsible rubber catheter has been tapered for easier penetration of the brain.‡ After the ventricle is entered and the stilette removed the cannula, if necessary, can be inserted its remaining length into the smallest ventricle without trauma. The other end of the rubber cannula is firmly threaded to a female Luer-Lok§ which in turn can be sutured to the scalp by means of a small metal flange† which we have fastened to the Luer-Lok and which is perforated at each end for the suture.

A metal plug¶ is inserted into the female Luer-Lok to close the system and can be removed at will for any additional procedure.

The parts of the cannula-catheter are shown in Fig. 1.

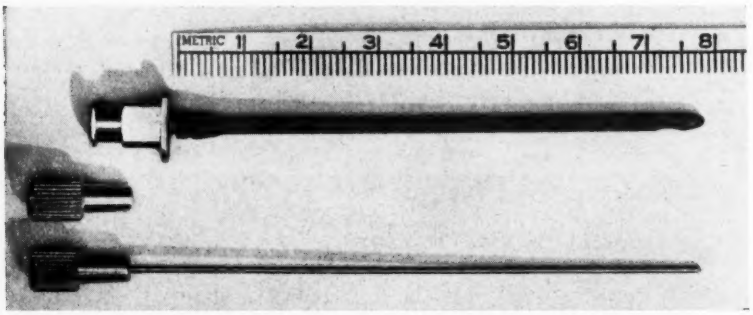

FIG. 1

Some advantages of this combined cannula and indwelling catheter are: (1) There is no danger of damage to the brain by any movement of the scalp as is present with a metal indwelling cannula. (2) Once the ventricle is reached the catheter is in place and it is not necessary to make a second insertion of a rubber or polyethylene catheter through the tract with danger of trauma to the brain, or through a metal cannula with the danger of dislodging the catheter when the metal cannula is pulled out. Further, there is always the problem of how to fasten these indwelling catheters to the scalp and to attach adapters to them. (3) The combined cannula-catheter can

* This cannula can be obtained from the George P. Pilling & Son Co., 3451 Walnut Street, Philadelphia 3, Penna.

† Made by the Temple University Hospital Machine Shop, Philadelphia, Penna.

‡ The Davol Rubber Company devised this catheter from their stock No. 8 French rubber catheter, modifying it to a wedge-shaped tip for easier penetration into the brain and hollow to the tip for snug fit of the stilette. The catheter can be cut to any length required.

§ Made by Becton, Dickinson Company, catalog number L/609.

¶ Made by Becton, Dickinson Company, catalog number 417A.

be left in situ for days without any unusual irritation. (4) During a craniotomy a ventricle can be tapped, the cannula left in place and the surrounding brain retracted for whatever approach is necessary (for example: transfrontal pituitary approach), without danger of brain damage from the catheter. (5) One can Luer-Lok any system for continuous ventricular injection, pressure estimation or drainage to this cannula.

There are some disadvantages in its use. It is not satisfactory for probing for tumor unless the stilette is kept in place. One must become accustomed to the "feel" of this cannula as compared to that of a metal one. When attempting to enter a small ventricle it is sometimes necessary to twirl or twist the cannula to free the holes in the end. Since the rubber is soft the cannula cannot be twisted unless the stilette is inserted. This is a minor inconvenience.

AN APPLICATING FORCEPS IN WHICH CLIPS CANNOT STICK OR JAM

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(Received for publication January 18, 1953)

All neurosurgeons at one time or another have been faced with the problem of clips sticking in the jaw of the clip applicator after the clip itself has been applied to an artery or a vein. The results of such an incident are often annoying, occasionally serious. Because of repeated incidents of this nature, it was felt that some means should be devised whereby the clips would either be dislodged from the grooves in the applicator jaw or prevented from jamming in these grooves at the time of application. To eliminate the possibility of faulty construction of clip forceps and variations in clip stock, applicators were carefully selected in which clips did

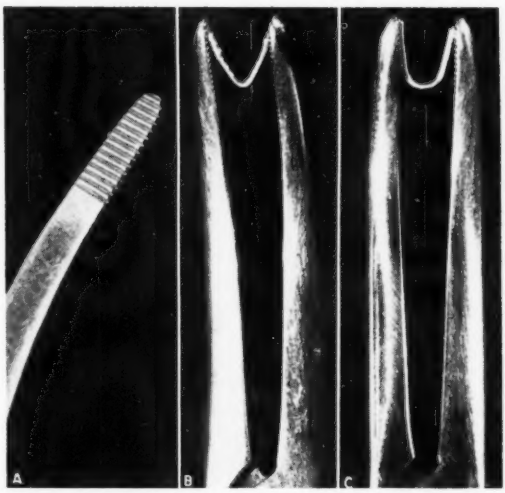

FIG. 1. (A) Detail of applicator jaw. (B and C) Standard "V" and "U" clips in the instrument.

not stick when tried at the factory. At the operating table, however, it was found that the sticking of the clips in the holders occurred just as frequently as with instruments selected at random. We felt, therefore, that the jamming of the clips was caused by bits of tissue that were wedged between the clip and the walls of the groove in the applicator tip.

To devise a means of dislodging the clip from the groove would have led to a more complicated type of applicator with consequent increase in cost and difficulty in maintenance. At the suggestion of Mr. Fred G. Todt., Sr.,* the problem was finally solved by a simple change in the jaw of the applicator whereby the longitudinal groove was entirely eliminated and rather heavy transverse serrations were substituted. This presents, as can be seen in Fig. 1, an essentially flat surface to the outer surfaces of the clip once the applicator jaws are closed, so that it is impossible for the clip to jam or stick to the jaw. This clip applicator is used with the standard McKenzie rod and Cushing clips or Duane "U"-clips.

This instrument has been used for several months under all conditions and has entirely eliminated the problem of clips jamming or catching in the jaws of the forceps. There is less tendency for the clips to become dislodged from the forceps in transfer from the nurse to the surgeon, and the operating room nurse has found that it is easier for her to pick clips off the McKenzie rod with this holder than with the standard holder.

Two minor difficulties that have been noted are a slight tendency for the clips to swing laterally and for the ends of the clips to become crossed if too much pressure is applied to the forceps. Both of these difficulties are solved by a very short period of experience with the applicator.†

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* Instrument designer, George P. Pilling & Son Company, 3451 Walnut Street, Philadelphia 4, Penna.

† This clip-applying forceps is available from George P. Pilling & Son Company, 3451 Walnut Street, Philadelphia 4, Penna.

AMERICAN BOARD OF NEUROLOGICAL SURGERY

For several years the American Board of Neurological Surgery has been somewhat concerned because some of the candidates who appear for examination do not seem to have had contact with an adequate amount of clinical material. Also, at the last examination, held in Chicago, Illinois, June 10–13, 1953, there was considerable concern on the part of the Board because in the examination that was given on general surgery many of the candidates showed little understanding of matters pertaining to the basic principles of general surgery, such as fluid and electrolyte balance, shock and hemorrhage, transfusion reactions, wound healing, etc.

This, together with other factors, has led the Board to set up officially a Residency Review Committee which will work in conjunction with the Council on Medical Education and Hospitals of the American Medical Association, and it is the intention of the Board that this Committee should actively review all residency training programs as soon as this is feasible. As a matter of policy, the Board has decided that an evaluation of each training program should include statistical data regarding the amount of clinical material that is available for residency training in the program. The Board has decided that a minimum amount of material necessary for the approval of a service should be the performance of 200 major neurosurgical operative procedures during the calendar year, and included in this list of 200 major neurosurgical procedures there must be at least 25 verified brain tumors. This amount of material is to be the requirement for each resident who is finishing training in a given year. In other words, if a residency training program is approved for three men for three years, this is interpreted by the Board as meaning that one man should begin and one man should complete his residency training program each year. If, on the other hand, there are three residents completing the training program each year, three times this volume of material should be available.

There has been considerable discussion by the Board regarding the problem of the actual surgery that is done by the resident. It does not seem feasible at the present time, however, to attempt to determine just what the division of work is between the resident staff and the visiting staff. The Board is also cognizant of the fact that in many private clinics little operative surgery is available for the resident staff.

As a part of this evaluation, the Board has decided that the list of operative cases should exclude what have been tentatively designated as minor procedures, and minor procedures are defined as including diagnostic burr holes, ventriculography, pneumoencephalography, angiography, alcohol injections, lumbar punctures, myelography and discography.

The Residency Review Committee will consist of three members from the Board and at the present time the Chairman, Dr. Grant, the Vice-Chairman, Dr. Spurling, and the Secretary, Dr. Furlow, have been designated as the Board representatives. There will be three members from the Council on Medical Education and Hospitals, one of whom will serve as the Executive Secretary, and this member will be from the Council office in Chicago. The other two members will be accredited neurosurgeons, and the appointments will be made from a list submitted by the Board. It is felt that the Committee will act in a more efficient manner than has been possible in the past. The Committee will meet as often as is necessary in Chicago and by full discussion will arrive at a decision which will be submitted to the Board for final approval. All of the clerical work will be done through the Council's office, and field

inspections will be made by representatives of the Council as has been done in the past. In addition, however, if there is any doubt, a regional evaluator will be designated by the Board to inspect the service and report to the Committee. The Committee will in turn report to the Board. Final approval will rest with the Board as it has in the past, but much correspondence will be saved, and the time for approval should be considerably shortened, since accurate reports can be submitted to the Board by the Committee at each meeting.

Examinations were given by the American Board of Neurological Surgery, June 10–13, 1953, in Chicago, Illinois, and the following were certified:

COOPER, IRVING S. 477 First Ave., New York 16, N. Y.
EDER, DAVID 127 North Madison Ave., Pasadena 1, Calif.
EPSTEIN, JOSEPH A. 36 East 38th St., New York, N. Y.
FISHER, ROBERT GEORGE Hitchcock Clinic, Hanover, N. H.
GOLDSTEIN, STANTON L. 307 First National Bank Bldg., Davenport, Iowa
GREEN, ROBERT EDMUND 153 Irvington Ave., South Orange, N. J.
HOWARTH, J. CORNALL Professional Bldg., North Main St., Orlando, Fla.
IRWIN, CARL WILLIAM 316 State St., Bangor, Me.
JACKSON, IRA JOEL University of Texas Medical School, Galveston, Tex.
KIMSEY, WARREN U. S. Army, Office of the Surgeon General, Washington, D. C.
KUHN, ROBERT ANTHONY University of Arkansas School of Medicine, Little Rock, Ark.
MIDENETS, HOWARD E. 31 Lincoln Park, Newark 2, N. J.
MURTAGH, FREDERICK 5603 Greene St., Philadelphia 44, Pa.
NILGES, RICHARD G. 700 North Michigan Ave., Chicago 11, Ill.
PADBERG, FRANK THOMAS 432 Waldon Bldg., Little Rock, Ark.
ROTH, DAVID B. 1005 Third National Bank Bldg., Dayton 2, Ohio
RUSSELL, JOHN R. Indiana University Medical Center, Indianapolis 7, Ind.
SCHWAB, JOHN E. Veterans Administration Hospital, Richmond 10, Va.
SCHWIDDE, JESS T. 307 Medical Arts Bldg., Great Falls, Mont.
SHAVER, MURTON R. Winter Veterans Administration Hospital, Topeka, Kan.
SMITH, JAMES RONALD 1607 Medical Arts Bldg., Fort Worth 2, Tex.
STREWLER, GORDON J. 1000 Medical Arts Bldg., Duluth 2, Minn.
SULLIVAN, BENJAMIN H. 736 Sixth St., South, St. Petersburg, Fla.
SUTTON, JOHN B. East Physicians & Surgeons Bldg., Shreveport, La.
TAHERI, ZIA EDIN (foreign certificate) Teheran, Iran
VAN DER WESTHAYSEN, PETER 504 Broadway, Gary, Ind.
WEAVER, SAMUEL W. 1205 North Broadway, Santa Ana, Calif.
WOODS, ROBERT P. 3617 West Sixth Ave., Topeka, Kan.
ZEALEAR, DAVID S. 1308 Olive Ave., Fresno, Calif.

The next examination will be given in New Haven, Connecticut, October 22–24, 1953.

EXPERIMENTAL INTERVERTEBRAL FUSION USING BASIC CALCIUM PHOSPHATE*

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(Received for publication February 2, 1953)

IN RECENT years, a desire to improve the results in the operative treatment of ruptured intervertebral discs has caused many orthopedic and a few neurological surgeons to combine disc removal with fusion of the spine by bone grafting on the lamina or dorsal spines.[2,8] Such a procedure has been considered unwarranted by most neurological surgeons.[1,9,11] They feel that adequate simple disc removal gives very satisfactory results. A more recent approach has been suggested by Cloward,[3] who uses a wedge-shaped bone graft driven into the evacuated intervertebral disc space to achieve "intervertebral fusion" and maintain a normal intervertebral distance. More recently Lindblom and Hultqvist[7] have proved that a protruded or ruptured nucleus pulposus may be completely absorbed, and thus advise only an anterolateral decompression of the disc space.[6]

The proponents of these several methods of treating herniated discs all claim good results. Barring gross discrepancies in the various reports, it seems possible that a common critical factor may exist in all these various operative methods to explain the comparable results claimed. The only operative step common to all these procedures is the evacuation of the intervertebral disc space at the level of the rupture. It has been generally recognized that this does result in at least a fibrous if not ultimately a bony union between the vertebrae,[5] though this has not been widely studied. There may be indirect evidence that such immobilization is an important factor since occasional patients who fail to respond adequately to the minimal disc removal show improvement following spinal fusion at a later date. Presumably if an early and complete intervertebral fusion could be achieved by a surgically benign procedure, the incidence of poor clinical results could be further minimized.

No extensive experimental or clinical evidence is available concerning the fibrous intervertebral fusion which supposedly follows complete removal of the intervertebral disc. The present investigation was proposed to determine two things: first, the effectiveness of the intervertebral fusion produced in monkeys following simple disc removal and curetting of the cartilaginous plates; and second, the effect on this fusion of synthetic

* This chemical was kindly supplied by Dr. E. P. Barrett, Mellon Institute, Pittsburgh.

† Chirurgisch Universitätsklinik, Graz, Austria.

hydroxyapatite introduced into the disc space at the initial operation. Synthetic hydroxyapatite is a crystalline basic calcium phosphate (BCP) the crystals of which show the same X-ray diffraction pattern and practically the same chemical and physical properties as bone. It is already known that when these crystals come in contact with the fresh bone surfaces the synthetic hydroxyapatite apparently acts as an "ossifiable medium" and thus promotes more rapid bone healing.[10]

MATERIALS AND METHODS

Eighteen monkeys, *Macaca mulatta*, 9 males and 9 females, were used in this project. Their weight ranged from 3.2 to 4.1 kg. Disc evacuation was done in 5 animals without introduction of BCP. In 2 of these, the nucleus pulposus alone was removed; in the remaining 3 monkeys, the disc space was completely evacuated and the cartilaginous plates were curetted down to bone. In 13 animals, the nucleus pulposus was removed and replaced by BCP paste; in 6 of these the cartilaginous plates were curetted. The paste was made by mixing 2 parts of powder with 1 part of saline. A single intervertebral space was evacuated in each animal though the level varied from L2 to L5. Motion of the normal lumbar spine was studied by X-ray in 3 animals.

After taking pre-operative lateral X-rays of the lumbar spine, the usual operative procedure was as follows:

The animals were anesthetized with nembutal, and the prevertebral space was reached either transperitoneally through a transverse abdominal incision or extraperitoneally by a paravertebral incision. The desired interspace was located and the annulus fibrosus was incised in the form of a U-shaped flap on its anterior or anterolateral surface. The jelly-like nucleus pulposus spontaneously protruded and was completely removed with a small spatula. The chondral surfaces of the adjacent vertebral bodies were curetted in certain cases, while in the others the cartilaginous plates were left intact. By means of a dental amalgam carrier, BCP paste was inserted into the evacuated interspace. The flap of the annulus fibrosus was then replaced and secured by a black silk suture. Muscles, peritoneum and skin were closed in a routine manner.

Postoperative lateral X-rays of the spine were taken immediately and repeated approximately every 2 weeks.

The physical activities of the animals were not restricted postoperatively. Animals in each group were sacrificed at about the 4th, 5th, and 6th month.

During the observation period, a needle was inserted under sterile conditions into the dorsal spine above and below the interspace evacuated at operation. These were used as X-ray markers to measure lumbar spine motion in hyperextension and flexion. A series of three such X-rays was taken in each animal prior to sacrifice. After sacrifice the animals were perfused with formalin, and the two vertebral bodies at the operative level were removed in one piece, fixed in formalin and decalcified. These speci-

mens were imbedded in low viscosity nitrocellulose, sections were cut at 10 microns and these then stained with hematoxylin and eosin. These slides were examined for histological evidence of intervertebral fusion, either fibrous or bony, and for the effect of the BCP on this process.

RESULTS

The histology of the normal intervertebral disc in the monkey is illustrated in Fig. 1. The vertebral bodies, the cartilaginous plates, and the

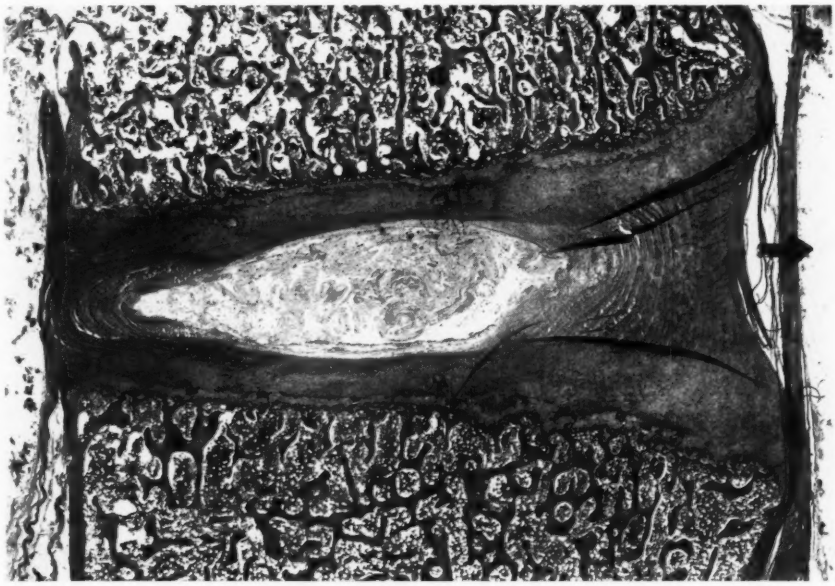

FIG. 1. Normal intervertebral disc of the monkey. (→is anterior)

annulus fibrosus surrounding the central nucleus pulposus are essentially the same as these structures in the human,[1] though the nucleus pulposus is grossly soft and gelatinous as in an immature human disc.[4] The X-rays of the normal intervertebral space in the monkey are likewise analogous to the human.

Complete removal of the normal nucleous pulposus without scraping of the cartilaginous plates results in fibrous intervertebral union as illustrated microscopically in Fig. 2. Here the nucleus pulposus has been replaced by fibrous tissue composed of proliferating fibroblasts and disorganized strands of collagen. The cartilaginous plates are seen to be intact. A striking difference is produced, however, when the cartilaginous plates are scraped at the time of operation. At the point of defect in the cartilaginous plates, actual proliferation of bone occurs which is, however, not sufficient to form a bony

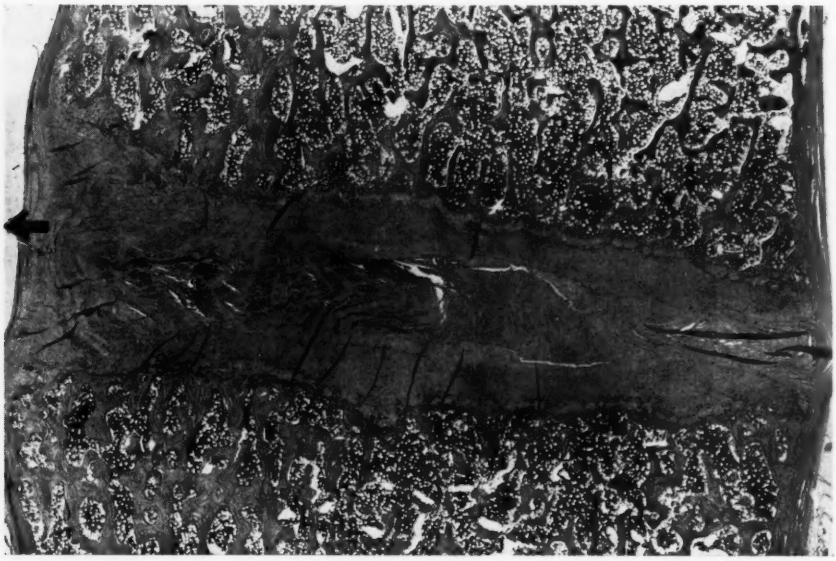

FIG. 2. Six months following simple disc evacuation.

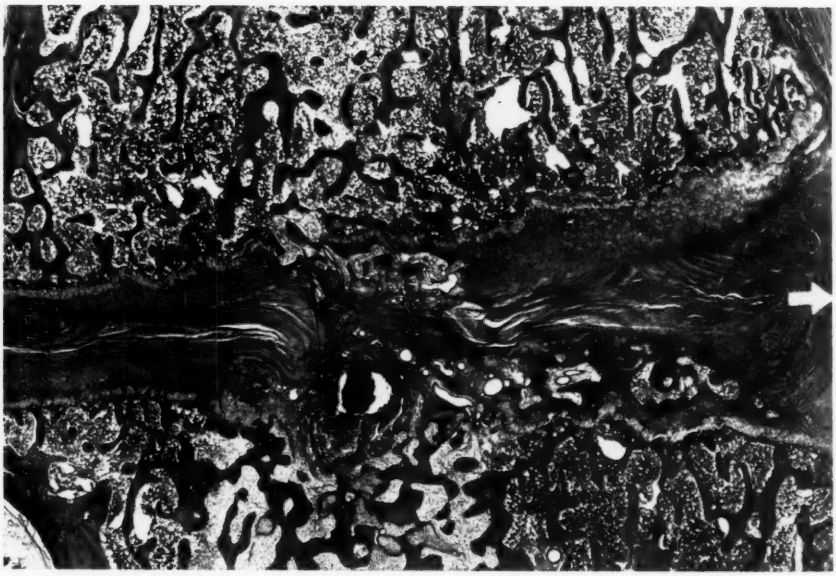

FIG. 3. Six months following disc evacuation with surgical disruption of the cartilage plates.

fusion even after 6 months (Fig. 3). The lack of bony fusion is also substantiated by the roentgenograms (Fig. 4), which show that no bony bridging is present in the interspace immediately below the marker.

Having established the normal pattern of response to disc removal in the monkey, it was then of interest to study the alterations in this pattern produced by the introduction of synthetic hydroxyapatite (BCP) into the disc space. In the cases in which the nucleus pulposus was completely removed without scraping the cartilaginous plates and the defect was filled with BCP, fibrous intervertebral union occurred (Fig. 5) similar to that seen in the control animal. Initially the evacuated disc space is full of BCP crystals which are gradually absorbed and replaced by proliferating fibroblasts and disorganized collagenous fibers. In Fig. 5 the result at 6 months shows that practically no BCP crystals remain, the cartilaginous plates are intact, and no bony proliferation has occurred. The fibrous union present is comparable to that seen in the control animals at the same time (Fig. 2). The X-ray shows no bony shadow in the interspace (Fig. 6).

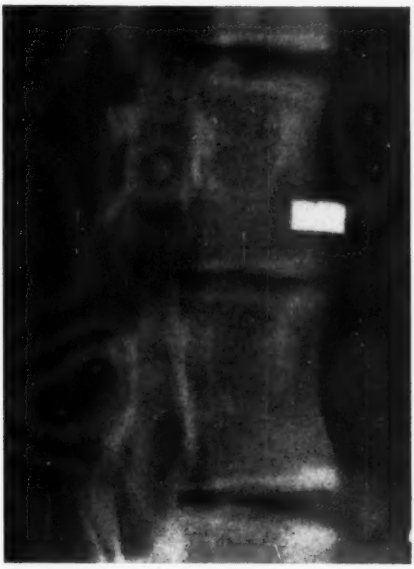

FIG. 4. X-ray of Fig. 3 showing bony invasion of interspace below marker without bony fusion.

A marked alteration of this pattern is produced when the continuity of the cartilaginous plates is disrupted at the time of operation. Under these circumstances, the intervertebral space initially is filled with BCP crystals in contact with cancellous bone at the points where the cartilage has been removed. At the end of 6 months, bony proliferation into the interspace has produced actual osseous bridging (Fig. 7). The X-ray substantiates this histological picture and at 6 months shows actual intervertebral calcification (Figs. 8 and 9). The initial cellular reaction around the crystals of BCP was essentially a foreign body reaction with the appearance of plasma cells and connective-tissue macrophages. No multinucleated giant cells were seen at any stage. It appeared that appositional bone formation occurred around the crystals with gradual replacement of these crystals by new bone formation. In the presence of osteoblasts, the BCP apparently acted as an ossifiable medium but in the absence of actively proliferating bone, there was no evidence that the BCP resulted in induced bone formation. By the end of 6 months, all the BCP had apparently been utilized (Fig. 7).

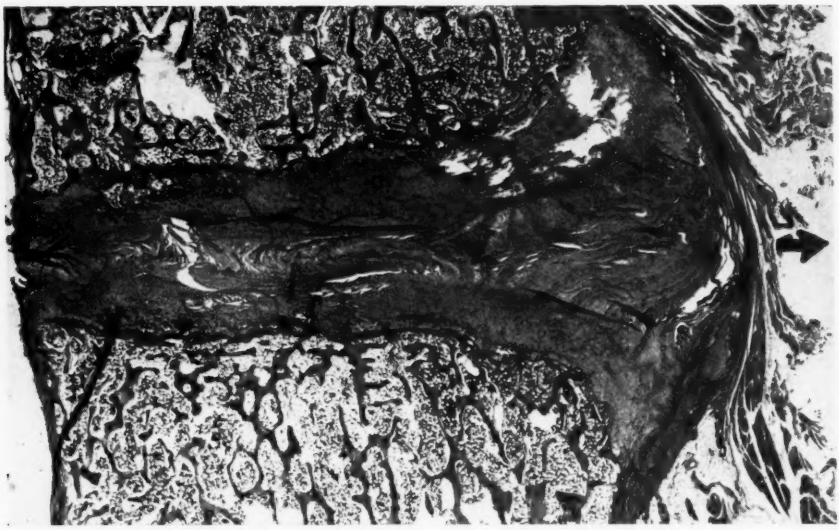

FIG. 5. Six months following evacuation of nucleus pulposus and introduction of BCP into disc space.

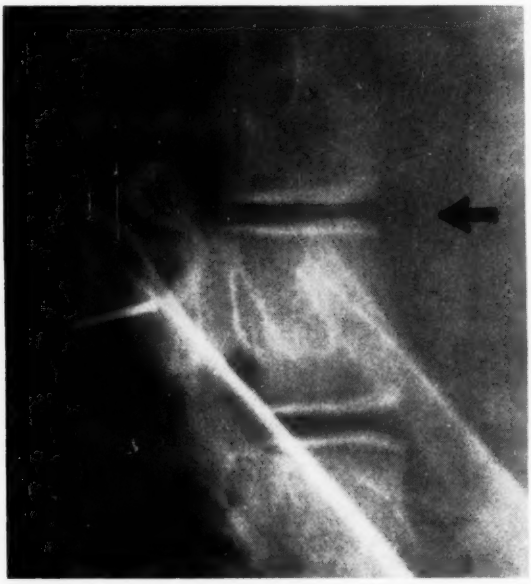

FIG. 6. X-ray of Fig. 5 showing no bony invasion of interspace.

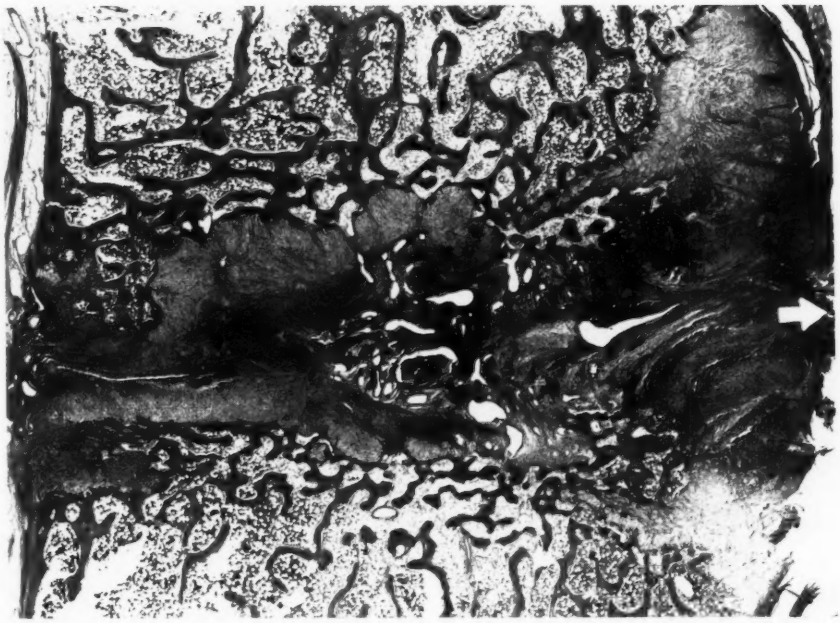

FIG. 7. Six months following disc evacuation, cartilage plate disruption, and introduction of BCP. Actual bony fusion is present.

The presence of the BCP appears to facilitate bony proliferation and bridging when it is in contact with cancellous bone as is seen by comparing Fig. 7 with Fig. 2. At the end of 3 months, there is histological evidence of bony proliferation when BCP is used but in the absence of the synthetic hydroxyapatite there is no evidence of bony proliferation even at 6 months. Thus it seems that the BCP increases both the total amount and speed of bony proliferation.

BCP powder was used in 3 cases, but in the remainder a paste was utilized since this permitted the introduction of a larger absolute amount of the hydroxyapatite into a fixed space. The immediate postoperative X-rays showed a denser shadow in the disc space when paste was used than when dry powder was used. This shadow usually persisted for 4 weeks and then disappeared rather rapidly. Another shadow appeared in the interspace at 2–4 months concomitant with the histological evidence of bony invasion of the disc space.

The presence of functional intervertebral fixation can best be determined by serial X-ray studies of the lumbar spine in which hyperextension and flexion films are accurately measured for intervertebral motion. Such motion can be determined by comparing the distance between the posterior ends of the marker needles in the dorsal spines during flexion and extension. In nor-

FIGS. 8 and 9. X-rays of Fig. 7. (*Left*) Note calcification within disc space at the marker (lateral film in anteflexion). (*Right*) Same in hyperextension showing no measurable change in the markers and indicating intervertebral fixation.

mal animals this motion was 5 to 11 mm. with an average excursion of 7 mm. No significant difference was found in the motion measured at levels from L2 through L5. In those cases in which the disc was evacuated and the cartilage plates were disrupted at operation, no motion was present at the operative level 6 months later. When this operative procedure was carried out and BCP introduced into the space as well, histological evidence of bony fusion and X-ray evidence of fixation was present at 3 months, indicating acceleration of this process by the BCP. In contrast, the cases in which simple evacuation of the nucleus pulposus was done, the average motion was 7 mm. at 6 months after operation, this being the same degree of motion seen in unoperated animals. When the nucleus pulposus was evacuated and the cavity filled with BCP, the average range of motion was 4 mm. at 6 months which may represent acceleration of the fibrous union.

DISCUSSION

It is evident that the normal intervertebral disc in the monkey is not equivalent to that in the adult human, although it does closely resemble the immature human disc. The results of this investigation are thus not entirely applicable to the human.

The fibrous intervertebral union following simple evacuation of the nucleus pulposus has been described by Compere and Keyes[4] in the dog. The fact that there is no limitation of back motion in the monkey following simple disc evacuation does not necessarily imply that no limitation will occur following disc removal in the human. This procedure in the human

involves removal of fibrocartilage, whereas in the monkey only the nucleus pulposus which has not been invaded by fibrocartilage is removed. When disruption of the cartilage plates is added to this operative procedure in the monkey, complete fixation and bony invasion occur, confirming previous observations in dogs and monkeys.[4,5] This finding is of interest since it is claimed that disruption of the cartilage plates is necessary to achieve the best clinical results in humans,[11] although actual bony bridging in the human has not been demonstrated. It has been widely assumed among neurosurgeons that fibrous union and at least partial fixation of the intervertebral joint occur following simple disc removal in the human. Extrapolating these experimental results to the human, one might anticipate that this fixation would occur and clinical studies are underway to demonstrate the presence or absence of such fixation. Disruption of the cartilaginous plates is necessary to obtain complete fixation in the monkey and it should thus be reemphasized that curettage of the cartilaginous plates in the human may well be an essential part of the operation if intervertebral fixation is necessary for complete and permanent relief of symptoms. This is especially true since such fixation may be the common critical factor in the various operative methods currently effective in the treatment of herniated disc.

Following simple disc evacuation in the monkey, introduction of a synthetic basic calcium phosphate (BCP) having the chemical and physical properties of bone did not produce bony intervertebral fusion. On the basis of previous studies by Ray and Ward,[10] it has been shown that BCP will act as an ossifiable medium only in the presence of proliferating osteoblasts. Following simple disc evacuation such osteoblasts are not available. However, X-ray studies of spine motion in these cases showed only one-half as much intervertebral motion as in those cases in which BCP was not introduced. Thus it would seem that although BCP can not stimulate bony intervertebral fusion in the absence of osteoblasts, it can promote a firmer fibrous union than otherwise occurs.

When disruption of the cartilaginous plates is added to simple disc evacuation, introduction of BCP then increases the rate and degree of bony intervertebral fusion. Curetting the cartilaginous plates provides a source of osteoblasts so that the BCP acts as an ossifiable medium,[10] thus increasing bony ingrowth into the intervertebral space. The result is earlier and more complete bony intervertebral fusion.

This experimental evidence points up certain factors of clinical significance. It is possible experimentally to produce complete intervertebral fusion by removal of the disc and curetting the cartilaginous plates. Such immobilization does not occur if the cartilage plates are left intact. If intervertebral fixation in the human is the common critical factor in various currently effective methods of treatment for herniated discs as we have postulated, it is then obvious that disruption of the cartilaginous plates by curettage is an essential part of the surgical procedure.

The experimental evidence indicates that even the fibrous union following simple disc removal is made more effective by the introduction of BCP

into the evacuated disc space. Such results would seem to warrant a clinical trial in humans. However, the surgical defect in the annulus fibrosus cannot be completely closed, and some of the inserted BCP inevitably extrudes itself. In the monkey, this was of no significance since the defect in the annulus was located anterolaterally in the retroperitoneal space. Such extrusions in the monkey have been demonstrated on several occasions by X-ray. However, the conventional posterolateral approach to the human disc places the annulus defect within the spinal canal beneath the nerve root, where extrusion of BCP would be undesirable. For that reason the use of BCP in the human is not contemplated unless the anterolateral, retroperitoneal approach suggested by Lindblom[6] is utilized.

SUMMARY

1. Simple disc removal in the monkey resulted in histological evidence of fibrous intervertebral union but caused no limitation of motion as demonstrated by X-ray studies.

2. Simple disc removal with replacement of the disc by basic calcium phosphate produced a more dense fibrous union with 50 per cent limitation of intervertebral motion.

3. Disc evacuation in combination with disruption of the cartilaginous plates resulted in bony invasion of the intervertebral space and complete intervertebral immobilization 6 months after operation.

4. Disc evacuation, disruption of the cartilaginous plates and introduction of BCP into the disc space increased the rate and degree of bony intervertebral fusion, as shown both histologically and by X-ray.

5. Possible clinical implications are discussed.

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EXPERIMENTAL USE OF A PREPARATION OF ETHYL DIIODOSTEARATE

AS A CONTRAST MEDIUM FOR DIAGNOSTIC CEREBRAL ANGIOGRAPHY

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CEREBRAL angiography has become in the past decade a valuable and frequently employed adjunct to diagnosis in disorders of the central nervous system. Of the radiopaque media currently employed, each has drawbacks peculiar to its own particular chemical and pharmacologic properties. In the attempt to produce a universally acceptable radiopaque compound for use in human angiographic studies, several agents have more recently been introduced for study.[3] One of these products, angiopac‡ (ethyl diiodostearate), was selected by us for study. Since the drug had as yet not been certified in the United States by the United States Food and Drug Administration for clinical use, preliminary investigation of its effects on animals seemed indicated.

Angiopac is a micro-ionized aqueous emulsion of ethyl diiodostearate. The product is milky in appearance, and the particles are considered to be 2 microns in diameter or one-fifth the size of a red blood corpuscle. According to Masy,[4] the compound is very opaque, painless on injection, well tolerated without side reactions, and rapidly eliminated by the kidneys.

The dose recommended by the manufacturer for cerebral angiography is 10 cc. per injection. Although maximal doses in man are not indicated, 40 cc. by intracarotid injection and 1 cc. per kg. by an intravenous route have been given with impunity, according to a brochure accompanying the drug.

Our interest in this product was with regard to four chief characteristics: (1) radiopacity, (2) toxicity, (3) irritant effect and persistence in tissues, such as muscle, where it might inadvertently be injected, and (4) elimination from the body.

METHOD

Carotid arteriography was carried out with angiopac in a series of 15 dogs and 5 cats. Only large animals (dogs weighing 20 kg. or more and cats weighing 4 to 5 kg. or more) were employed. All injections were made by open

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‡ The material used in this study was supplied by the Union Chimique de Belge in Bruxelles.

technic; anesthesia was used for all dogs except 1 whose carotid artery had been exteriorized previously. Ether, pentobarbital sodium (nembutal) or thiopental sodium (pentothal sodium) was used in the dosage that had heretofore been found satisfactory for routine operations on animals at the Institute of Experimental Medicine, Mayo Foundation. As nearly as possible, conditions identical to those of human angiographic procedures were simulated. Simultaneous polygraphic recordings of pulse rate, blood pressure, respiration and heart action were obtained from a cannula in the femoral artery, a pneumograph, and an electrocardiograph.

The dose of angiopac ranged from 0.5 cc. to 10 cc. per injection. No animal received a total of more than 20 cc. in any series of repeated injections. Two dogs received the injections into the pectoral muscle.

RESULTS

Three dogs died as the direct result of the injection of angiopac into the carotid system. Two dogs required artificial respiration and termination of the procedure. No cats died as a result of injections.

The typical reaction to the injection of angiopac in dogs was a fall in blood pressure (Fig. 1).

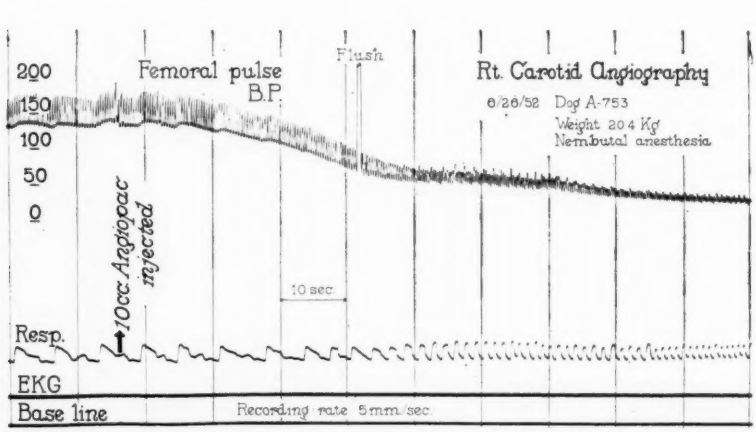

FIG. 1. Polygraphic recording to show a typical response to a 10 cc. injection of angiopac in a dog weighing 20.4 kg.

Spacing the injections at intervals of 5 minutes or more, the only dose of angiopac consistently tolerated by dogs was 3 cc. or less. The smaller amounts, 1 to 2.5 cc. were tolerated best. Larger injections, 8 to 10 cc., tended to cause critical symptoms and death. Death was usually caused by respiratory failure. Similar amounts of iodopyracet (diodrast) did not have the above-mentioned effects.

The radiographic contrast was excellent and, we believe, superior to that

of diodrast, particularly in showing the detail of small vessels. Some have attributed this to the fact that less vascular spasm is caused by angiopac (Fig. 2).

Angiopac was purposely injected into the pectoral muscle of 2 dogs. This was followed by an intense reaction at the site of injection, manifested by swelling, erythema, and tenderness to palpation. Microscopically the reaction was evidenced by an intense polymorphonuclear infiltration (Fig. 3). There was radiographic evidence of dye retained in the tissues 3 months after local injection (Fig. 4).

Two dogs and 1 cat were anesthetized and killed 1 to 2 days following injection of angiopac into the carotid artery. Roentgenograms of the heads

FIG. 2. Radiographic contrast with angiopac, showing detail of small vessels.

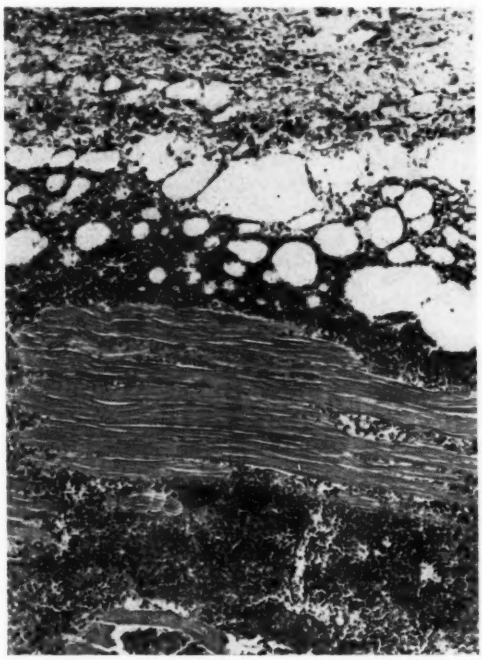

FIG. 3. Section showing intense cellular reaction to angiopac injected into muscle (hematoxylin and eosin, ×100).

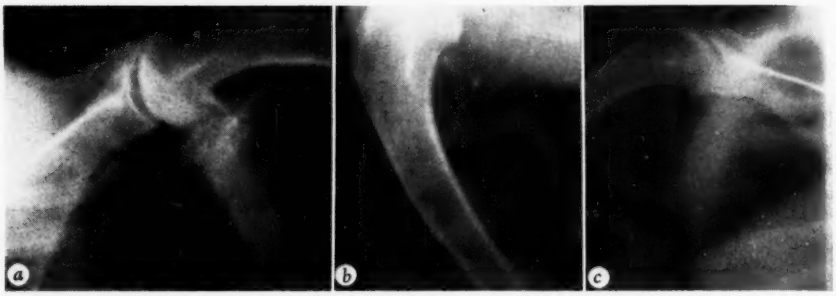

FIG. 4. Dorsal and ventral roentgenographic views of the right pectoral region (dog A-626): (*a*) 1 week, (*b*) 7 weeks, and (*c*) 11 weeks after injection of 2 cc. of angiopac.

and bodies of these dogs and histologic examination of organs such as the kidney, brain and liver disclosed no evidence of damage or of residual angiopac.

CONCLUSION

It has been suggested that the unusual response of dogs to the intracarotid injection of angiopac and the lack of effect on cats may be ascribable to an allergic response to the solvent (a derivative of polyoxyethylene sorbital) used in the manufacture of angiopac. Some support of this is given by Krantz and co-workers.[1,2] On the basis of our experience in animals, and because it is known that occasionally such reactions may occur in man, we believe that the incidence of side reactions in man might be greater than we presently encounter with 35 per cent diodrast. Hence we have hesitated to employ angiopac clinically.

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MESENCEPHALIC TRACTOTOMY FOR PAIN

EXPERIENCE WITH SIX CASES

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THE convergence of the central trigeminal and spinothalamic tracts in the pons and mesencephalon offers an opportunity for division of the pain pathways from the opposite half of the body. Dogliotti[1] (1938) first carried out this procedure in the upper pons. Walker[6] in 1942 showed that the same result could be obtained in a less hazardous manner by tractotomy at the level of the mesencephalon, and studied the physiological effects of tractotomy at this level in considerable detail.

Walker's[6] patients lost the ability to appreciate painful and thermal stimuli throughout the entire side of the body contralateral to the lesion. Superficial and deep pain were immediately relieved. Touch sensation was not impaired when tested with cotton wool, although a definite increase in the tactile threshold was demonstrable with von Frey hairs. The division of the lateral lemniscus that overlies the pain pathways produced no subjective disturbance of hearing; however, audiometer tests revealed impairment of the appreciation of high tones. There may be temporary minimal weakness of the contralateral lower extremity but there was no disturbance of proprioceptive sensation or coordination.

In view of Walker's[6] findings we operated on 6 patients to satisfy ourselves on the feasibility and results of the procedure. The essential features in the 6 patients selected for operation are shown in Table 1.

Cases 4 and 5 might well have been treated by other surgical procedures but were included in the series in an effort to establish the relief of facial pain

TABLE 1

Case	Age	Diagnosis	Site of Intractable Pain
1	63	Traumatic disarticulation right arm —22 yrs. previously.	Shoulder stump.
2	51	Cutaneous hyperpathia following intercostal neurectomy.	Right paravertebral region from T3–T8.
3	47	Bronchogenic carcinoma.	Left shoulder, supraclavicular fossa, left arm, and left upper chest.
4	37	Carcinoma, left maxillary antrum.	Left face.
5	49	Carcinoma, right nasopharynx.	Right ear, face and neck.
6	63	Bronchogenic carcinoma, left.	Left shoulder and arm.

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by section of the secondary trigeminal pathways at the level of the mesencephalon. Our experience with these 6 patients will be detailed under the following headings: (1) Operative approach. (2) Physiological results. (3) Relief of pain. (4) Postoperative dysesthesia.

1. *Operative Approach.* All of our patients have been operated upon in the sitting position. This has been satisfactory and in our opinion made the operation easier. A small occipital bone flap is turned down on the side opposite the pain, so as to expose the lateral sinus below and the sagittal sinus medially. The occipital lobe is retracted upwards and laterally. It was necessary in all but one case to coagulate and divide one or two occipital veins as they entered the lateral part of the lateral sinus. No veins enter the mesial portion of the lateral sinus or the posterior part of the sagittal sinus. The tentorium is incised about one inch from and in line with the straight sinus. The posterolateral aspect of the midbrain may then be exposed by tearing the arachnoid overlying the cisterna ambiens. In our experience there have been no undue difficulties in identifying the landmarks on this aspect of the midbrain as shown in Bancroft and Pilcher's[5] book.

As shown by Walker[7] the pain pathways at this level lie just deep to the brachium of the inferior colliculus and are somewhat intermingled with its deep fibres. There is a topographical arrangement, so that the pain fibres from the lower extremity lie dorsolaterally and those from the head and neck ventromedially.

The tractotomy incision is made with a cordotomy knife, and is 5 mm. in depth. It extends from the lateral sulcus, medially and slightly upwards across the brachium of the inferior colliculus to the lower pole of the superior colliculus. Selective partial division of the mesencephalic pain pathways is feasible and has been carried out by Walker.[6] Although no deliberate attempt was made to do this in our series one patient obtained a level of analgesia up to the umbilicus only. In this patient, at a second operation, under local anaesthesia the tractotomy incision was lengthened, ventromedially. There was no corresponding rise in the cutaneous sensory level. It was necessary to make a separate tractotomy incision 3 mm. rostral to the former in order to obtain complete hemianalgesia. No pain was experienced when the incision was made into the pain pathways at this level.

2. *Physiological Results.* In an effort to corroborate the physiological effects of the procedure as described by Walker,[6] careful pre-operative studies were carried out, including perimetry and tests of vestibular and cochlear function. Special attention was paid to the examination of the sensory system. No attempt was made to determine accurately the thresholds of touch and pain with von Frey thorns and hairs. Each patient was carefully examined with respect to the subjective equality of the response evoked on each side of the body in regard to cutaneous, deep and discriminative sensation.

All 6 patients showed a loss of pain and temperature sensation throughout the opposite side of the body and head. The sensory disturbances extended to the midline.

Deep pain was abolished on the involved side with the exception of the testicle. Deep pressure in that half of the abdomen, on the muscle bellies, Achilles tendon, subcutaneous bones, brachial plexus, ulnar and supra-orbital nerves, was without response from the patients. The same was true of deep pressure on the lateral wall of the rectum and vagina. One patient consented to have ureteral sensation investigated. Dermaskin bags were threaded up each ureter through a cystoscope and inflated at regular intervals throughout the course of the ureter. On the analgesic side, the ureter was insensitive in its upper two-thirds only. The lower one-third and the other ureter had normal sensation. Distension of the bladder produced a normal response.

This situation persisted for days or a few weeks, then a change occurred; stimulation now provoked an unusual abnormal pain response which will be detailed in Section 4.

Subjectively, touch sensation was consistently diminished on the analgesic side, although to a minimal degree, for with the eyes closed the patient was always able to recognize at once the lightest stimulus with cotton wool. This was at variance with Walker's[6] observations in which the lowered touch threshold could be demonstrated only with von Frey hairs. The central pathways for touch are widely dispersed and it was to be expected that a small incision in the midbrain would not affect this sensation to any marked degree.

By standard clinical tests vibration and position sense were not altered by the operation with one exception, in which there was evidence of damage to the cerebral peduncle as well as to the medial lemniscus. As Walker[6] stated, there was no evidence in our cases to suggest that discriminative sensation was impaired in any way.

The vestibular responses were not impaired when tested postoperatively. Walker[6] described this in his cases and in a personal communication to one of us (McKenzie) has stated that bilateral mesencephalic tractotomy results in total loss of hearing. None of the patients complained of disturbance of hearing. In 2 of the 3 patients tested postoperative audiograms showed persistent decrease in the perception of high tones.

Taste sensation was not affected either subjectively or objectively in the 2 patients examined.

Five of the patients showed a temporary complete homonymous hemianopsia in the contralateral field of vision. The production of the field defect seemed to be related as much to the retraction of the occipital lobe as to the division of the occipital veins. In one patient the hemianopsia was not present until the third postoperative day. Another had no field defect although a large vein was divided. The last patient, who was operated upon twice, had no field defect following the first procedure in which two veins were divided. Following the second operation, during which no further veins were divided, he had a complete homonymous hemianopsia for 5 days. Each patient had full visual fields to perimetric examination from 5 to 7 days after operation. The hemianopsia probably was derived from a combination of pressure on

the optic radiation caused by the retraction and temporary impairment of the cortical circulation, the result of venous interruption. Two patients complained of difficulty in reading anything but large print for 3 or 4 weeks, but this was not permanent.

3. *Relief of Pain.* In the early postoperative period all 6 patients were free of their intractable pain with the exception of one, who after completion of tractotomy at a second operation obtained complete relief. Headache was restricted to the side of the head, with no sensory disturbance. Walker[6] found that even the headache produced by the intravenous injection of histamine was chiefly confined to one side. Some days later, a spontaneous burning dysesthesia developed on the analgesic side in 3 of the 6 patients. This dysesthesia was considered to be more intolerable than the original pain. In addition an unusual abnormal pain response to sensory stimulation developed in all 6 patients. This will now be discussed in more detail, because we feel that it is a very serious postoperative complication.

4. *Postoperative Dysesthesia.* After a period varying from 3 or 4 days to 2 weeks after the operation a new response developed to pin prick, deep pressure or thermal stimuli in all 6 patients. In 1 patient even the lightest touch provoked a violent response. Upon stimulation the patients complained of a deep, diffuse, poorly localized pain associated with strong withdrawal and grimacing. They described it as agonizing and miserable—an entirely new experience for them. In addition to this, spontaneous burning pain on the side with disturbed sensibility developed in 3 patients. In 2 this dysesthesia, although generalized over that half of the body, was worse in the region of their previous pain. One man who had had a unilateral prefrontal lobotomy prior to the tractotomy stated that he was nearly driven insane by this dysesthesia. In another patient unilateral prefrontal lobotomy failed to relieve the dysesthesia although her reaction to it was dulled somewhat. Both these patients stated that this spontaneous pain was much more intolerable than the pain for which they were operated upon. In the third patient, who had been operated upon for intractable pain in the face, burning pain developed in the same region. Intramedullary tractotomy 3 months later gave him complete relief.

The nature of this intolerable pain similar to pain in the thalamic syndrome has been the subject of some thought by several authors.

Head and Holmes[2] suggested that the pain associated with a thalamic lesion is the result of interruption of corticothalamic fibres, the function of which is to inhibit the reaction of the thalamus to afferent impulses arriving at the thalamus.

Kendall[3] based his theory on the assumption that painful sensations are of two distinct types, each conducted centrally by separate sets of fibres—one fast, the other slow. Normally the delayed disagreeable sensation conducted by the slow fibres is blocked by a thalamic inhibitory mechanism set up by the arrival at the thalamus of impulses conducted by the fast fibres. If however, the fast fibres are destroyed, the inhibitory process fails and the

poorly localized, diffuse, disagreeable pain is felt. Kendall also felt that the total amount of damage to the pathways conducting these afferent impulses must not be very extensive. In his case[3] in which there was complete loss of temperature appreciation there was no over-reaction to hot or cold objects. Each of our patients had complete loss of pain and temperature sensation on the side of the dysesthesia.

The dysesthesia in our patients following discrete lesions of the central pain pathways just prior to their entry into the thalamus cannot, of course, be explained by the theory of Head and Holmes,[2] who postulated interruption of corticothalamic fibres.

The recent work of Magoun *et al.*[4] suggests that the great afferent systems give off collaterals into the reticular formation as they pass up the brainstem. There is as yet no anatomical verification of Magoun's work. One of us (Drake) suggests that lesions of the pain tracts low in the brainstem or in the spinal cord would interrupt the central pathway provided by these collaterals. If, however, the lesion is above the origin of the collaterals—even though it may be complete—a secondary route via the reticular formation would provide a means by which the slower impulses might reach consciousness. Such a pathway would account for the delay in perception and diffuseness of the pain.

We have seen this same abnormal response to stimulation but to a minor degree in at least 3 patients who had the spinothalamic tract divided high in the first cervical segment. We cannot recall seeing this response following section of the tract in the upper thoracic region.

CONCLUSIONS

From our experience with 6 cases of mesencephalic tractotomy the following conclusions are drawn:

1. The operation is technically quite feasible and the landmarks are definite. A good physiological result with loss of appreciation of pain and temperature over the whole of the opposite half of the body is obtained. There are no severe neurological sequelae, such as profound disturbance of balance or paralysis. However this excellent temporary result was nullified in our cases by the development of intolerable, spontaneous dysesthesia in 3 out of 6 patients, coupled with a new disagreeable response to stimuli in all 6 patieuts. This postoperative dysesthesia has profoundly influenced our thoughts about the operation; so much so that we are unlikely to carry out the procedure again. An anatomical explanation is suggested to explain this postoperative dysesthesia in Section 4 of this report.

2. We feel that the technique and knowledge of spinothalamic tractotomy in the upper cervical and lower medullary region will gradually develop and replace mesencephalic tractotomy for the relief of shoulder and arm pain.

3. Pain in the face and upper neck secondary to carcinoma is best relieved by section of the descending tract of the 5th nerve, the glossopharyngeal and upper filament of the vagus and the upper cervical dorsal roots.

This calls for a smaller exposure than that necessary for section of the 5th root at the pons. In addition the patient is spared the additional misery of complete anaesthesia in the tongue and mouth area.

The authors wish to thank Doctor E. H. Botterell for permission to include Case 6.

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EFFECT OF PREVIOUS SENSORY LOSS ON THE APPEARANCE OF HERPES SIMPLEX

FOLLOWING TRIGEMINAL SENSORY ROOT SECTION*

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In a study of patients with trigeminal neuralgia in whom sensory root section had been performed via the extradural, subtemporal, approach, the incidence of postoperative herpes simplex infection in the denervated areas was surprisingly high, 16 of 17 patients in the initial series having had this complication.[3] Since this report, 28 of 30 patients have shown typical homolateral herpetic lesions postoperatively, a total of 44 of 47 cases (93.6 per cent). It was established that this clinical eruption was without doubt caused by herpes simplex virus.** It was also postulated that the herpes simplex following trigeminal sensory root section was produced by reactivation of herpes simplex virus latent in the skin and mucous membranes, rather than by migration peripherally from the ganglion.[3]

In considering the mechanism of this type of virus activation, certain facts deserve consideration: (1) Herpetic lesions are not associated with peripheral interruption of the trigeminal nerve, nor with section of the intramedullary portions of its sensory root.‡[6,11] (2) Herpetic lesions are infrequent following direct injection of alcohol into the Gasserian ganglion (and consequent ganglion cell destruction).§ (3) Herpetic lesions appear more frequently when the posterior root section is closest to the ganglion, as compared to root section near the entry of the nerve into the pons; injury, but not death, to the ganglion cells appears to be the common denominator. (4) Alteration of sensation in the 2nd or 3rd divisions of the trigeminal nerve prior to subsequent sensory root section, prevents the appearance of herpetic

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** Virus studies were performed by Dr. E. D. Kilbourne (formerly Fellow, Rockefeller Institute for Medical Research, now Associate Professor of Medicine and Director, Division of Infectious Diseases, Tulane University School of Medicine) and Dr. S. Ellison (School of Dentistry, and Department of Bacteriology, College of Physicians and Surgeons, Columbia University).

‡ Sjöqvist[12] described a patient in whom intramedullary tractotomy following trigeminal sensory root section via the temporal approach produced herpes, despite the presence of cutaneous anesthesia of the face. Sweet[13] likewise has noted the appearance of herpes following intramedullary tractotomy.

§ In reported cases of alcohol injection of the Gasserian ganglion, herpes rarely developed in patients with excellent anesthesia. This is taken to indicate almost complete destruction of ganglion cells in those cases. When herpes occurred, ganglion cells were considered to be injured, but not destroyed.

lesions that usually occur following the operative procedure.* Previous intact sensation is suggested as one of the prerequisites of virus activation by trigeminal sensory root section, although this may not be true of intramedullary tractotomy. The following data amplify this thesis.

MATERIALS AND METHODS

In a series of 47 consecutive trigeminal operations (posterior root section via middle fossa approach), 8 patients had had prior injections or operative procedures, consisting of peripheral alcohol injection, peripheral nerve avulsion, or partial posterior root section. The subsequent posterior root section was performed for the relief of recurrent or persistent pain. The appearance of postoperative herpetic lesions was noted and their distribution recorded.

RESULTS

In 5 of 8 patients, herpetic lesions followed the secondary operative procedure, these appearing in 48 to 72 hours. The lesions were homolateral to the operated side, but did not appear in any instance in the previously denervated areas. In 3 patients in whom lesions did not appear, neutralizing antibody or a positive complement fixation test for herpes simplex virus was obtained from pre-operative serum. The clinical data which follow are summarized in Fig. 1.

Case 6 had had a previous peripheral alcohol injection (done elsewhere) in the right mental foramen (Fig. 2, 1) which had resulted in a cutaneous slough of the cheek with a large scar and permanent anesthesia of the skin of the chin and lower lip, with the exception of the angle of the right side of the mouth and the adjacent lateral portion of the lower lip. Following differential section of the 2nd and 3rd divisions of the sensory root, herpes did not appear in the anesthetic area, but did appear in the portion of the 3rd division that had remained normally innervated, as well as in the 2nd division.

Case 3 had had a previous left infra-orbital neurectomy (Fig. 2, 2) resulting in total anesthesia in the distribution of that nerve. A sensory root differential section was then performed, with excellent anesthesia in the 2nd and 3rd divisions. Herpes appeared only on the hard palate on the left, with no cutaneous lesions in the 2nd division; there was a marked eruption on the lower lip and chin on the left.

Case 5 had had a previously attempted sensory root section of the 3rd division intracranially. Actually, the outer third of the ganglion itself and probably the mandibular nerve just prior to its entrance into the ganglion were severed (Fig. 2, 3) as indicated by the extremely sharp line of cutaneous anesthesia at the border of the 2nd division. Here a subsequent sensory root 2nd and 3rd division section resulted in herpes of the upper lip, but none in the area previously denervated.

* Furlow[8] has injected one or two drops of absolute alcohol into the mandibular and/or maxillary nerve intracranially just peripheral to the ganglion prior to closure following trigeminal sensory root section. With both nerves injected, he felt that the incidence of postoperative herpes was negligible with one or the other of the nerves injected, herpes, if it appeared, was always present in the distribution of the nerve that had not been injected.

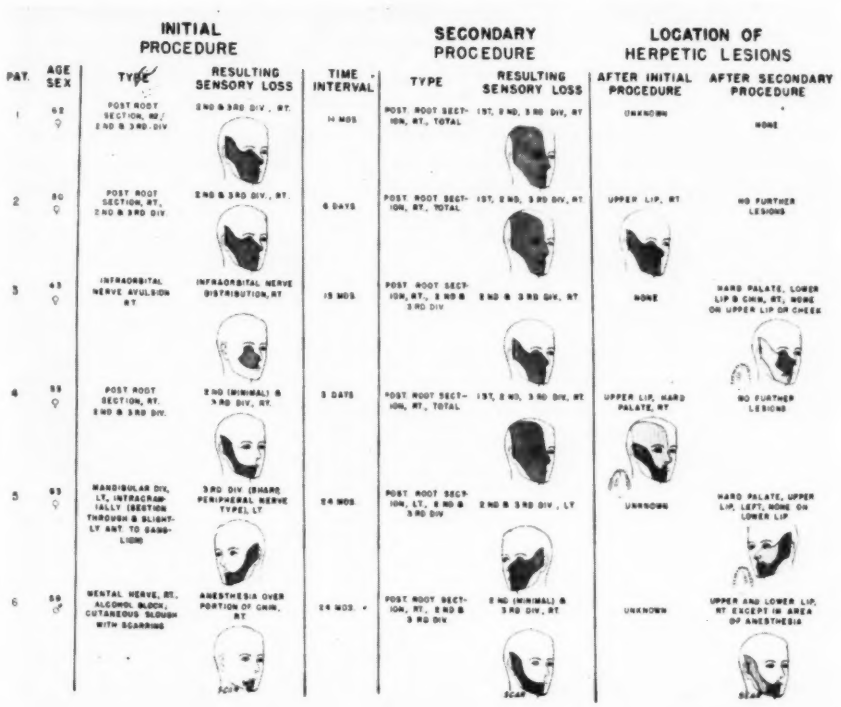
INITIAL PROCEDURE
SECONDARY PROCEDURE
LOCATION OF HERPETIC LESIONS
PAT.
AGE SEX
TYPE
RESULTING SENSORY LOSS
TIME INTERVAL
TYPE
RESULTING SENSORY LOSS
AFTER INITIAL PROCEDURE
AFTER SECONDARY PROCEDURE
1
62 ♀
POST ROOT SECTION, RT., 2ND & 3RD DIV
2ND & 3RD DIV., RT.
14 MOS
POST ROOT SECTION, RT., TOTAL
1ST, 2ND, 3RD DIV, RT
UNKNOWN
NONE
2
80 ♀
POST ROOT SECTION, RT., 2ND & 3RD DIV
2ND & 3RD DIV., RT.
6 DAYS
POST ROOT SECTION, RT., TOTAL
1ST, 2ND, 3RD DIV, RT.
UPPER LIP, RT
NO FURTHER LESIONS
3
43 ♀
INFRAORBITAL NERVE AVULSION RT.
INFRAORBITAL NERVE DISTRIBUTION, RT
15 MOS.
POST ROOT SECTION, RT., 2 ND & 3 RD DIV
2ND & 3RD DIV., RT.
NONE
HARD PALATE, LOWER LIP & CHIN, RT., NONE ON UPPER LIP OR CHEEK
4
58 ♀
POST ROOT SECTION, RT. 2ND & 3RD DIV.
2 ND (MINIMAL) & 3 RD DIV., RT.
3 DAYS
POST ROOT SECTION, RT., TOTAL
1ST, 2 ND, 3 RD DIV., RT.
UPPER LIP, HARD PALATE, RT
NO FURTHER LESIONS
5
63 ♀
MANDIBULAR DIV., LT., INTRACRANIALLY (SECTION THROUGH & SLIGHTLY ANT. TO GASSERIAN)
3 RD DIV. (SHARP PERIPHERAL NERVE TYPE) LT
24 MOS.
POST ROOT SECTION, LT., 2 ND & 3 RD DIV
2 ND & 3 RD DIV., LT
UNKNOWN
HARD PALATE, UPPER LIP, LEFT, NONE ON LOWER LIP
6
59 ♂
MENTAL NERVE, RT., ALCOHOL BLOCK, CUTANEOUS SLOUGH WITH SCARRING
ANESTHESIA OVER PORTION OF CHIN, RT
24 MOS.
POST ROOT SECTION, RT., 2 ND & 3 RD DIV
2 ND (MINIMAL) & 3 RD DIV., RT
UNKNOWN
UPPER AND LOWER LIP, RT EXCEPT IN AREA OF ANESTHESIA

FIG. 1. Effect of previous sensory loss on location of herpetic lesions. (Cases 7 and 8 are not represented.)

Case 1 (Case 6 of the previous series[3]) had had a previous 2nd and 3rd division sensory root section (Fig. 2, 4) and subsequently, 11 months later, a total sensory root section (Fig. 2, 5). No herpes appeared postoperatively. Neutralizing antibody to herpes simplex virus was demonstrated in the patient's serum.

Case 7 had had a previous left total posterior root section. Pain in the right side of the face was then noted, treated by alcohol blocks of the infra-orbital and mandibular nerves, resulting in hypesthesia and hypalgesia in the sensory distribution of these nerves. A differential left 2nd and 3rd division posterior root section was performed, without the subsequent development of herpes. Neutralizing antibody was present in the pre-operative serum.

Cases 2 and 4 had originally 2nd and 3rd division sensory root sections, which were followed by the appearance of herpes. Secondary operation, namely total sensory root section, was performed 6 and 3 days respectively after the initial procedure, resulting in no further herpes, the original lesions having resolved in Case 2 and at their height in Case 4. Pertinent conclusions cannot be drawn from these latter two procedures.

DISCUSSION

These clinical cases clearly show that herpes simplex appearing after trigeminal sensory root section did not occur in skin or mucosal areas that had been previously denervated. The failure of herpetic lesions to appear in those cases in which neutralizing antibody was present preoperatively (indicating latent virus or previous infection) is thus explained by the preoperative anesthesia in the areas in which the virus was usually latent. How-

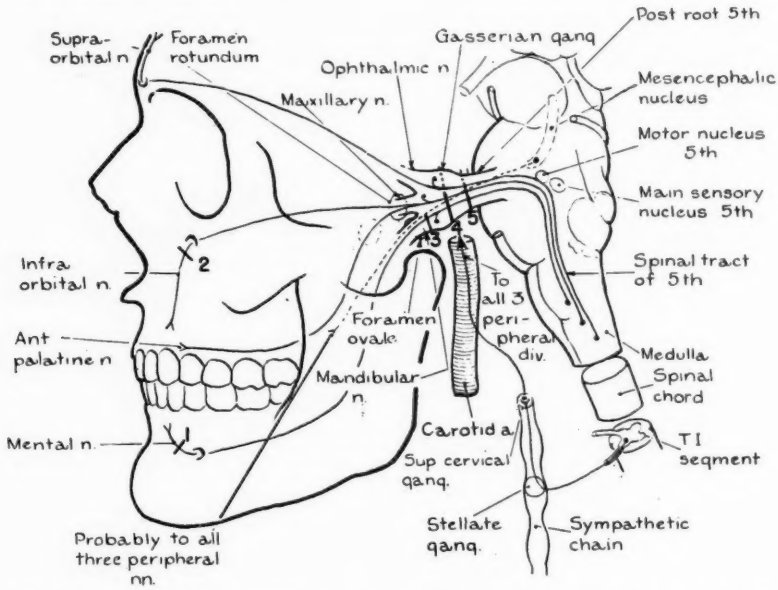

FIG. 2. Anatomical pathways severed by various surgical procedures. (Modified after A. Brodal, 1948; and R. C. Truex and C. E. Kellner, 1948.)

ever, the precise relationship of sensory root section or injury of the ganglion to the production of herpetic lesions has not been elucidated.

The neural pathways severed by the different surgical procedures are indicated in Fig. 2. The general somatic afferent fibers are those with cells of origin in both the Gasserian ganglion and mesencephalic nucleus in the pons.[5] Section of the trigeminal sensory root or of a peripheral nerve branch will cause degeneration of the distal portion of the mesencephalic pathway. On the other hand, section of the sensory root will not cause degeneration in the peripheral axons whose cells in the Gasserian ganglion are intact, although the centripetal axons entering the spinal tract of the 5th nerve will degenerate and will not regenerate.[10] In addition, following alcohol injection of the Gasserian ganglion transsynaptic degeneration of the cells in the main sensory and spinal tract nuclei of the 5th nerve has been described, but not de-

generation in the mesencephalic nucleus.[10] There is capacity for regeneration of the ganglion cell pathway peripheral to the ganglion, as evidenced by recovery of sensation following alcohol injection; data concerning regeneration of the mesencephalic pathway are not available. The efferent sympathetic fibers arising from the secondary neuron in the stellate or superior cervical ganglion comprise the third pathway. These join the peripheral nerve at or immediately distal to the Gasserian ganglion, coursing with the three peripheral divisions of the trigeminal nerve. Clinical evidence of regeneration is likewise available for this pathway.

The most likely theory of virus activation by trigeminal sensory root section is one that invokes stimulation of the end-organ in the skin by some process resulting from the surgical procedure. Since herpes did not occur in the cases under discussion unless the ganglion had been apparently injured by surgery,* injury to the ganglion cells without actual cell death may be postulated as producing irritative or stimulatory phenomena. Adrian[1,2] in his work on the injury potential, has shown that injury to a sensory nerve may produce stimulatory phenomena. The fact that herpes will not be produced if the peripheral pathway has been severed or rendered functionally useless by alcohol injection, implies that the irritative phenomena cannot be transferred along the nerve from ganglion to end-organ unless the neural pathway is intact.

Sympathetic overactivity or unopposed activity as the cause of the stimulatory changes in the skin might be postulated, since these fibers enter the 5th nerve complex distal to the ganglion and would be still intact. The fact that injury potentials do not develop in sympathetic fibers[1] and that clinical signs of sympathetic overactivity are absent, makes it unlikely that activation of virus is a sympathetic nervous system phenomenon.

The available data may be summarized in a highly speculative theory of virus activation for these particular conditions. Section of the sensory root, especially if close to the ganglion, may lead to injury of ganglion cells and secondarily to increased irritability of the sensory nerve associated with repetitive synchronous discharges. These impulses pass centrifugally in an antidromic fashion, producing changes in the skin, resulting in concomitant increased excitability within the local sensory unit. When the neural stimuli cease, the cutaneous changes are already well established and persist, leading to an increased local "host susceptibility"[7] to a virus already latent in the affected area, the virus in turn being activated. The striking histochemical similarity between the changes in the sensory nerve cell and in the cells of the skin secondary to the effects of repetitive stimulation[4] are of interest in this regard in that these two structures are linked by an intact and conducting axon. When such a link has been broken, as above, the changes in

* A unilateral herpetic eruption has been observed frequently by the author and others[9,13] following routine craniotomies in which no approach was made to the Gasserian ganglion or sensory root. The mechanism of this phenomenon appears to be different from that in the present study.

the nerve cell cannot affect the host cell in the skin and the virus remains latent.

SUMMARY

1. In 44 of 47 patients (93.6 per cent) with trigeminal neuralgia in whom sensory root section had been performed via the extradural, subtemporal approach, a typical homolateral herpes simplex muco-cutaneous eruption was noted in the postoperative period.

2. In 5 of 8 patients subjected to a secondary operative procedure herpetic lesions likewise appeared postoperatively. However, in no instance did they appear within the sensory area denervated by the original procedure.

3. The neural pathways involved and the anatomical changes noted therein following the surgical procedure are briefly discussed.

4. It is postulated that damage to the ganglion cells secondary to the surgical procedure caused increased irritability of the respective sensory nerve fibers. This resulted in changes in the skin, in which the herpes simplex virus is latent; reactivation of virus then occurred. Previous interruption of the sensory pathways involved prevented this sequence of events.

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POSTOPERATIVE INFLAMMATORY DISEASE OF LUMBAR DISCS*

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THE surgical treatment in certain cases of sciatica by removal of the protruding portion of a lumbar disc has become a fairly uniform procedure. Criteria for operation, the technical procedure, and the functional end-results are much the same from one medical center to another. In contrast to the banal appearance of this group as a whole, I will describe 3 patients whose courses deviated a long way from the beaten track. These are instances of presumed infection that developed within the disc after operation. They are described as presumed infection because culture of the causative organism was not obtained in any of them, although a psoas abscess was drained in one, and purulent fluid drained from the disc of another. The 3 cases are dissimilar in several ways, but parallel in respect to their long morbidity, their evidence of severe toxemia, and their end-result.

It is necessary to relate that in my experience with the surgery of lumbar discs (about 300 operations) there has been no instance of major postoperative infection arising in the soft tissues. I have not enjoyed the same immunity from gross soft-tissue infections in cases in which a combined disc and fusion operation was performed. But I am speaking of cases in which the disc alone was disturbed, and in these, healing of the wound has been the rule.

Acute infection of intervertebral discs is usually classified with osteomyelitis of the vertebrae. In 1940, Ghormley, Bickel and Dickson[5] of the Mayo Clinic reported 20 cases in which a diagnosis of infectious spondylitis had been made. They were focussing attention on a special group of cases in which there was very little osteomyelitis of vertebral bodies, but evidence of severe infection within a disc. Three of their patients showed clinical evidence of irritation of the central nervous system—a point in common with my cases. None was related to operation.‡

Certain anatomical and pathological features of intervertebral discs influence the course of infection. Within the normal disc after the third dec-

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Submission for publication was postponed in expectation of a more illuminating discussion from some neurosurgeon of greater experience with disc surgery. This tardy appearance may stimulate such a report.

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‡ Since writing this paper brief references to postoperative infections have been noted in two publications by Armstrong.[1,2]

ade, there are no blood vessels or lymphatics.[3] This situation is altered following injury. When there has been a rupture through the posterior margin of a disc the process of healing involves an intrusion of blood vessels into the disc along with the invading fibrous tissue.[4] If the rupture has been very gross the protruding lump of fibrocartilage may have to be pulled out at operation. Then there will be even greater vascularization of the interior of the disc. In either case, a blood-borne infection might find a satisfactory locale for activity. In the patient who has had operation an adjacent extradural infection would find an easy path of entry. The poorly healed hole in the posterior spinal ligament and fibrous cover of the disc is like a weak trap door.

With these pathological postulates in mind, I will describe briefly the clinical stories of 3 middle-aged British Columbians—a Finn, a Swede, and a Czecho-Slovak. They are all grateful for surgical relief from severe sciatica, but badly shaken by the morbid events that followed.

Case 1. G.H., Finnish workman, aged 46.

In July 1947 the patient presented a typical clinical syndrome of protruded intervertebral disc with sciatica, which had been initiated by injury. The signs were characteristic of involvement of the left 1st sacral root. Myelogram disclosed a rather indefinite defect on the left side at L5-S1. Disc interspaces were normal.

Operation. A small, firm protrusion was found at L5-S1 and pulled out as one chunk. The interior of the disc was not curetted.

Course. His initial postoperative progress was uneventful. Temperature was normal by the 3rd day. On this day a sore throat and cough developed, which continued for a week. He was out of bed on the 10th day and left hospital 3 weeks after operation.

Three weeks later he returned, looking miserable and complaining of severe headache with vomiting, which had troubled him for about 3 days. He was re-admitted to hospital. His neck was very stiff and he had bilateral Kernig sign. Cisternal puncture was done; the CSF contained 525 WBC, 44 per cent polymorphs, 56 per cent lymphocytes, and 115 mg. protein per cc.

He was started on large doses of penicillin. CSF obtained by lumbar puncture 3 days after admission showed 1150 WBC, 28 per cent polymorphs, 72 per cent lymphocytes, and 200 mg. protein. Successive lumbar punctures disclosed a gradual improvement in the CSF which coincided with a gradual clearing up of his meningitic signs and symptoms. His temperature had remained normal and his pulse rate in the 60's from the beginning. Two months after admission the CSF cell count was 33, with 90 per cent lymphocytes, and 105 mg. protein.

As the headache cleared up increasingly severe low backache developed, with pain radiating to both legs. He was discharged from hospital after 3 months, still complaining of pain in his lower back.

A month later he looked very miserable, so shaky that he had to lie down to take off his low-back belt, depressed and unnerved. He returned to his home in a small town up the B.C. coast. Up there, under the direction of a physiotherapist he made a slow, but excellent recovery. He returned to his regular work, about 1 year after the original operation.

Two years later he was still working steadily with only occasional stiffness in his back after a hard day. There was moderate limitation of flexion of the lower

back. Roentgenograms showed apparent solid fusion at the lumbosacral level with generalized calcification and considerable new bone production anteriorly.

Case 2. N.B., Swedish workman, aged 50.

He was first examined on Jan. 7, 1949, complaining of sciatica in the left leg, with typical story and clinical signs of a protruded disc. There was no pre-operative myelogram. AP and lateral X-ray films before operation showed nothing remarkable.

1st Operation. The interlaminar spaces on the left side between L4 and S1 were explored. At the L4–L5 level a rupture and protrusion of the disc was disclosed. The usual stringy chunk of fibrocartilage was pulled out and further soft cartilage was removed from within the disc with curette.

Course. We expected a good recovery, but he did very poorly from the start. He complained of headache for about 4 weeks, but after the 1st week the headaches were overshadowed by severe pains in his lower back. For about 5 weeks he had a daily rise in temperature from normal to 100° or 101° and a persistent, high sedimentation rate.

He was a very thin man before operation and during the course of the illness the loss of weight became extreme. Six weeks after operation myelography was performed. The CSF showed no cells but a protein of 50 mg. There was a large filling defect at the L4–L5 level, occupying the anterior part of the canal above and below the level of the disc. There appeared to be some destruction of bone on the inferior surface of the body of L4.

2nd Operation. On entering the disc, a thin purulent fluid poured out. A large amount of soft degenerated cartilage was removed. This cartilage was heavily infiltrated with polymorphs, lymphocytes and some plasma cells. No organisms, tuberculous or otherwise, were found on smear and culture showed no growth.

Course. Two months after the 2nd operation, roentgenograms showed some destruction of the inferior portion of the body of L4, and slight narrowing of the L4-L5 disc space. New bone formation was beginning to bridge lateral margins of the space. After the 3rd month his temperature remained normal. Four months after the 2nd operation X-ray films demonstrated further consolidation of new bone around the L4–L5 space. His general condition remained so poor that it was not until the end of 6 months that he was fit to leave hospital. Two months later he was getting along well and returned to his home in the country.

Case 3. J.M., Czecho-Slovak farmer, aged 44.

The patient was first seen in July, 1946, complaining of recurrent right-sided sciatica of 6 years' duration. The history was typical of protruded disc. The spinal fluid was normal. Plain X-ray films appeared normal. Myelography demonstrated filling defects at both the L3–L4 and L4–L5 levels.

1st Operation. Both levels were explored. A characteristic protrusion of disc was encountered at the L4–L5 level. The protruding loose mass was pulled out and further cartilage was removed from within the disc by curette.

Course. Recovery was rapid and uneventful.

Three years later (March, 1949) he returned complaining of sciatica in the opposite (left) leg, which had troubled him for 3 months. He was grossly disabled. Again the symptoms and signs were typical of disc protrusion. X-ray films demonstrated that the L4–L5 interspace had been well maintained. There were no gross adjacent degenerative changes.

He was admitted to hospital for myelography. To our surprise the spinal fluid

removed prior to this test showed a cell count of 77, with 30 per cent polymorphs, 70 per cent lymphocytes and protein of 50 mg. The myelogram showed some irregularities, but nothing that was considered clearly diagnostic. Blood counts were normal but sedimentation rate was elevated to 38 mm./hr. (Westgren). During the subsequent 3 weeks spinal fluid obtained by cisternal puncture was checked three times. The cell count remained in the 60's with the same relative percentage (30) of polymorphs. There was a daily temperature rise to 100°. He had no headache or stiff neck. The sciatic symptoms gradually diminished and he was discharged to continue rest at home.

He returned 3 months later complaining of return of severe pain in his lower back and left leg and was re-admitted to hospital. He had lost 30 lbs. in weight since the development of symptoms, and was having occasional severe night sweats. There was marked restriction of forward bending and bilateral hamstring spasm. He had an irregular low-grade fever. The spinal fluid was normal but WBC was 10,000 and sedimentation rate 84 mm./hr.

2nd Operation. The L4–L5 disc was re-explored. On the left side—the side of his radiating pain—the disc did not bulge and was not opened. On the right side, from which a protrusion had been removed 3 years before, the former hole was re-entered. No loose chunks of fibrocartilage were found within the disc and nothing was removed. There was no indication of infection.

Course. Pain in his back and low-grade fever and high sedimentation rate continued. Massive doses of penicillin and later large doses of streptomycin had no apparent effect. Two months after admission there was swelling of the operative area.

3rd Operation. Exploration revealed a small, deep abscess which did not seem to extend deeper than the lamina. Smear and culture were negative.

Course. After a further month the wound was healed. He was free of pain and had no fever but still showed a sedimentation rate around 50 mm. He was discharged from hospital with a plaster body cast, 4 months after re-admission.

He returned to hospital 2 months later (9 months after 2nd admission). Now he had a large, left psoas abscess and clearcut evidence by X-ray of a destructive process of the vertebral bodies adjacent to the L4–L5 disc which was markedly narrowed. Pus aspirated from the psoas abscess was negative on smear and culture. The abscess was drained and healed. He eventually returned home, 1 year after the delayed recurrence of sciatica. He was symptom-free, though still wearing a Taylor brace.*

DISCUSSION

One might enquire where the infection originated, or how it was introduced. Was it blood-borne, introduced by myelography, by spinal anesthesia or by operator? The data do not support a detailed argument. It is my opinion that in all 3 cases the infection was introduced at operation and was primarily a localized infection in the extradural space. That would account for the phase of meningeal irritation. In the first 2 cases the pathway into the disc was wide open from recent operation. The third case, with the 3-year interval, might be explained by a chronic, silent extradural infection

* One year later he returned for observation. Clinical examination was negative. He did not show up for recheck X-ray films.

which was stirred up by the second injury, and then provided with a fresh pathway of entry by the second operation. It is noteworthy that in Case 1, in which the curette had not been introduced into the disc at operation, there was no apparent acute infection of the adjacent vertebrae, whereas in the other 2 cases, in which the cartilage plates had been scraped, there was evidence of invasion of infection into the vertebral bodies.

CONCLUSIONS

This has been a report about a statistically insignificant group of cases. They illustrate the severity and chronicity of infection that may occur within a lumbar disc after an operation. The development of a clinical state that resembles tuberculous meningitis suggests the onset of this complication. My experience has not indicated how to treat it, except by expectant, conservative measures.

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THROMBO-ENDARTERECTOMY

FOR THROMBOSIS OF THE INTERNAL CAROTID ARTERY IN THE NECK*

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WITHIN the past few years several reports concerning thrombosis of the internal carotid artery in the neck have appeared in the literature. The increase in the reported incidence of primary thrombosis of the cervical portion of the internal carotid artery has generally been attributed to the development and the prevalent use of cerebral angiography.[3,4] The early recognition of thrombosis of the internal carotid artery in the neck on clinical grounds alone is almost impossible, since the symptoms and signs are not distinctive, but may occur with cerebral thrombosis, brain tumor, and other space-occupying lesions.[1,2]

CASE REPORT

#60580. J.T., a 52-year-old white male, was admitted on Jan. 15, 1953. On January 12, the patient had stated that he felt ill. He complained of headache ano refused to eat. Shortly thereafter members of his family noticed that he had weakness of the right side of the face and the right extremities. These symptoms and signs cleared in 1 day. Two days later he began to talk "nonsense" and complained of inability to see to the right.

Examination. He was a well developed, well nourished acutely ill right-handed white male whose lips appeared to be slightly cyanotic, although he was not in respiratory distress. He was indifferent to his surroundings. The temperature was 100.2°; pulse rate 92; respiratory rate 16; and B.P. 140/75. The peripheral pulses in all four extremities were present and equal, and there was no striking evidence of generalized arteriosclerosis or of a systemic vascular disease. There was a marked sensory aphasia with perseveration. Right-left dissociation and finger agnosia were present.He answered questions either with jargon or "Holy God." Occasionally he gave appropriate simple answers. He was able to understand and follow simple commands. Complex commands were inadequately carried out. Motor function was good bilaterally. The right abdominal reflexes were absent and there was a positive Babinski sign on the right. All other reflexes were normal. On bilateral simultaneous stimulation there was extinction of sensation on the right side of the body and face. The eyegrounds showed early papilledema. A gross right homonymous hemianopsia was present. Accurate testing of the visual fields could not be carried out because of the patient's inability to cooperate.

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* Presented before the New York Society of Neurosurgery, February 17, 1953.

Analyses of the urine, spinal fluid, and blood, including hematologic, chemical, and serologic studies, disclosed no abnormalities. Roentgenograms of the skull showed a calcified pineal body located in the midline. An initial diagnosis of thrombosis of a branch of the left middle cerebral artery was entertained.

Course. During the 1st week the condition of the patient was relatively stationary. Four days after admission roentgenograms of the skull showed a 2 to 3 mm. shift of the pineal body to the right. Three days later the deviation was estimated to be 6 mm. (Fig. 1). An EEG disclosed left-sided electrical abnormality, with no definite localization. In view of these developments the diagnosis was changed to that of a left temporoparietal tumor.

Angiography. On Jan. 23, 1953, four injections of 35 per cent diodrast (8 cc. each) were made into the left common carotid artery by the percutaneous route. Films in the anteroposterior and left lateral projections showed filling only of the external carotid artery and its branches.

During the next 2 days the patient exhibited transitory periods of increasing drowsiness, accompanied by irregular respirations of 12–14 a minute. There was no change in his neurological status. It was felt by one of us (K.J.S) that papilledema was not present, and that the most likely diagnosis was thrombosis of the left internal carotid artery in the neck. The pineal shift was explained on the basis of unilateral cerebral edema secondary to anoxia associated with decreased blood flow through the left hemisphere.

FIG. 1. Pineal shift visible in roentgenogram of skull.

Angiography was repeated on January 26, after insertion of a 17-gauge needle into the left common carotid artery. A single injection of 10 cc. of 35 per cent diodrast was made and a single exposure in the left lateral projection was made on a 10×14″ film. This revealed satisfactory filling of the external carotid circulation. The flow of diodrast through the internal carotid artery was arrested at a point approximately 1.5 cm. beyond the bifurcation (Fig. 2). This confirmed the diagnosis of thrombosis of the internal carotid artery.

Because of the possibility of attacking the lesion surgically, it was advisable to determine the adequacy of the cerebral circulation through the circle of Willis. Cerebral angiography by way of the right common carotid artery was done, despite the hazard entailed. Serial exposures in the lateral and anteroposterior projections were made during four injections of 7 cc. of 35 per cent diodrast. Films revealed filling of the right and left anterior and middle cerebral arteries. The intracranial portion of the left internal carotid artery filled in a retrograde manner for a distance of approximately 6–8 mm. (Fig. 3).

Operation, Jan. 28, 1953. Through an incision parallel to the anterior border of the left sternocleidomastoid muscle, from the tip of the mastoid process to the

FIG. 2. Obstruction of cervical portion of internal carotid artery, in lateral angiogram.

level of the cricoid cartilage, the common carotid artery and its two branches were exposed. The carotid sinus was infiltrated with 1 per cent procaine. Approximately 7 cm. of the internal carotid artery were mobilized. At a point 2 cm. distal to the carotid bifurcation there was an abrupt change in the appearance of the internal carotid artery. The caliber of the vessel was about two-thirds that of the proximal portion and a definite cyanotic hue was seen (Fig. 4). These changes were present throughout all of the distal segment visualized. Loops of umbilical tape were placed around the external carotid and the most distal portion of the internal carotid artery, and the common carotid artery was occluded with a Potts multi-toothed clamp. A 2-cm. longitudinal incision was made at the point of transition in the affected vessel, and a thrombus partially extruded itself through the opening. A piece of clot with adherent intima was removed, exposing a markedly sclerotic lumen. Partial opening of the artery clamp permitted a flow of blood which forced another piece of clot proximal to the incision through it. A catheter (#10 Fr.) with tip removed was passed distally toward the skull. With suction applied to the catheter at all times it was possible to remove additional thrombus. A total of 7 cm. of clot was removed (Fig. 5). The traction tape on the internal carotid artery was then released, but a retrograde flow of blood could not be obtained. Several attempts to remove the intracranial portion of the clot were unsuccessful. Because

FIG. 3. Partial retrograde filling of intracranial portion of left internal carotid artery, via right carotid angiography.

of the danger of dislodging clot in the intracranial portion of the internal carotid artery when the common carotid artery clamp was removed, the vessel was occluded by transfixion ligatures and a 2 cm. portion was removed (Fig. 6). The wound was closed in layers with interrupted black silk sutures.

(*For Figure 4, see page 479*)

FIG. 5. Clots removed from internal carotid artery (×1.5).

Postoperative Course. The patient's condition was satisfactory. He showed no ill effects from the operative procedure. There has been some improvement in the aphasia, but this is not attributed to the surgical treatment, being merely the expected course of his disorder.

DISCUSSION

This case presented a difficult problem in diagnosis. Repeated x-ray studies of the skull, while giving a time record of progressive shift of the pineal, were misleading in giving information that is usually conclusive evidence of an intracranial mass. The right homonymous hemianopsia, aphasia, and right hemisensory defect localized the disease process in the temporal and parietal lobes. Despite the pineal shift it was still felt that the most likely lesion accounting for the clinical picture was thrombosis of the internal carotid artery in the neck. The pineal shift was considered to be the result of cerebral anoxia. It may be assumed that ventriculography, if it had been done, would have shown a ventricular shift to the right.

FIG. 6. Resected sclerotic segment of vessel (×3).

With these facts in mind, and with the realization that this is only a single case, one may conjecture concerning how many negative intracranial explorations may have been carried out in cases of internal carotid artery thrombosis. The accepted indications for surgical intervention may be present, including pineal shift, clinical localization, and, probably, shift of the ventricles, and yet all be secondary to this vascular occlusion. Angiography utilizing the right internal carotid artery was carried out with full awareness of the risks involved. However, it was felt that confirmation of the presence of a thrombus in the left internal carotid artery and determination of the status of the cerebral circulation would be of value in deciding whether or not the lesion could be attacked surgically.

The presence of a thrombus in the left internal carotid artery in this patient presented a challenge concerning treatment. Ligation and resection of a portion of the artery has been done by others to reduce the tendency toward cerebral arterial spasm. In this case, it was elected to expose the common, external and internal carotid arteries and, if possible, remove the thrombus. Certain dangers to the patient existed, chiefly the induction of cerebral arterial spasm and the dislodging of a portion of clot which might have entered the middle or anterior cerebral arteries. The first was controlled by giving the patient papaverine and by injecting procaine around the bifurcation of the common carotid artery as soon as it was exposed. The second was minimized by applying suction to the intra-arterial catheter. This, supplemented by the flow of blood from the opposite carotid artery pushing downward on the clot, was considered an adequate means of preventing embolization. The left cerebral circulation appeared to be adequate as demonstrated by angiography.

Depending on the location of the thrombus and success in removing it,

FIG. 4. Probe points to transition zone at site of thrombus in internal carotid artery; tape is around external carotid artery.

a choice of one of three procedures was possible. A thrombus well fixed near the carotid bifurcation, with removable clot distal to it, could have been by-passed by anastomosing the external carotid artery to the patent portion of the affected vessel. If the thrombus could have been completely removed, then ligation of the external carotid artery might have enhanced the flow of blood to the brain. Thrombo-endarterectomy could have been carried out as in comparable segmental lesions in the extremities. In this case the distal portion of the clot could not be removed. Thus we were left with no other choice but to ligate and remove a portion of the artery. It was considered unsafe to allow blood under full arterial pressure to flow against a possible loose clot and throw it into the cerebral circulation.

The failure of filling of the cerebral circulation during angiography, with evidence of patency of the external carotid artery, can be assumed to be presumptive evidence of thrombosis of the internal carotid artery. Normally the major portion of the radiopaque medium flows toward the brain. Unless the cervical portion of the carotid system is seen on the films an important phase of the study is missed. Therefore, it is necessary to make every attempt to visualize the carotid bifurcation. This is where a serialograph with small films is inadequate. With this deficiency in mind, a single supplementary large film exposed during the injection of the contrast medium will give all the information needed. It is advisable to do this routinely in all cases of suspected vascular lesions. Partial occlusion low in the internal carotid artery may permit passage of the substance, showing a normal intracerebral circulation, and missing the lesion entirely. Thus a large film can be exposed initially, and if a carotid lesion is found further injections would not be necessary. This procedure was followed during the second study of the left carotid arteries. Another case of internal carotid artery thrombosis had been previously diagnosed by one of us (K.J.S.) and verified on a large film after serial studies showed filling only of the external carotid artery and its branches.

It is conceivable that, if the diagnosis in this case had been made earlier, it might have been possible to remove the clot completely. The sensitivity of the brain to even short periods of anoxia mitigates against the probability of achieving total rehabilitation. Extension of the thrombotic process to the intracranial portion of the artery is an obstacle that has not yet been overcome. One may anticipate improvement in some of these cases by this procedure.

SUMMARY

A case of thrombosis of the internal carotid artery in the neck simulating a brain tumor is presented.

The lesion was diagnosed clinically and verified by angiography.

An attempted surgical removal of a clot in the internal carotid artery is described.

Although the clot could not be completely removed, it has been demonstrated that such a procedure may be feasible.

A by-passing arterial anastomosis or thrombo-endarterectomy should be

successful when the occlusion is localized to the cervical portion of the internal carotid artery. Early diagnosis and removal of a localized thrombus can probably result in reducing cerebral damage and prevent further insult caused by vascular insufficiency.

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BILATERAL CINGULO-TRACTOTOMY

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Men ought to know that from brain and from brain only arise our pleasures, joys, laughter, and jests as well as our sorrows, pains, griefs, and tears. Through it in particular we think, see, hear and distinguish the ugly from the beautiful, the bad from the good. All that we suffer comes from the brain. That is why I hold that the brain is the most powerful organ of the body.

HIPPOCRATES

THE purpose of the present study has been twofold: first, to develop a comparatively safe, simple, selective and effective operation that will eliminate or diminish as much as possible a patient's psychosis and/or pain, at the same time leaving intact most if not all of his personality, and second, to apply this operation to psychotic patients, especially early and untreated schizophrenics, as the first and not the last resort. In developing this method there have been taken into consideration the neurosurgical virtues of "open" technic (actual visualization, selective section and complete haemostasis) and the nonsurgical merits of the "blind"[4,5] procedure (conservatism, speed and simplicity). In short, bilateral cingulo-tractotomy is Poppen's[13,14] bimedial lobotomy made more selective and less destructive.

The material to be presented is unique in that it is Eastern. Eastern mentality and outlook differ in many respects from the Western. Jung's "collective unconsciousness" helps to explain some of the Easterners' views. On the whole they are more primitive, dependent and fatalistic.

For the successes Allah is praised.
For the failures doctor is blamed.

ANATOMICO-PHYSIOLOGICAL CONSIDERATIONS

After the epoch-making operation of Moniz and Lima,[11] Papez[12] advanced the hypothesis that the hippocampus and the structures closely connected with it, such as the cingulate gyrus, orbital surface of the frontal lobes, and temporal cortex, and the association fibre tracts act as a neuronal substrate for emotions. Since then neuroanatomists and neurosurgeons have concentrated on this area.

Le Gros Clark[1–3] showed how the prefrontal lobes are connected with the thalamus and the hypothalamus by so many fibres passing in both directions that this entire system may be considered as functioning as an integrated whole. . . . The activities of the nervous complex consisting of prefrontal lobes, thalamus and hypothalamus are correlated with some of the higher intellectual activities, the personality, emotional affects and forms of be-

haviour of social significance. Fulton and his co-workers[6–8] divided the forepart of the brain into two major divisions: (1) The "visceral brain," which includes midline structures, and corresponds roughly to Papez's neuronal substrate for emotions; and (2) the so-called "neocortex," which occupies the lateral surface of the cerebral hemispheres and is most prominently developed in man. The visceral brain, which operates through the hypothalamus, is concerned with the integration of emotional responses, whereas the neocortex of the lateral surface is concerned with the integration of learning and the more highly intellectual functions. This "visceral brain" is the present-day target in psychosurgical operations.

There is an area of forebrain (Brodmann's Area 32) that does not belong to the visceral brain, but it is unavoidably destroyed by lobotomy. This

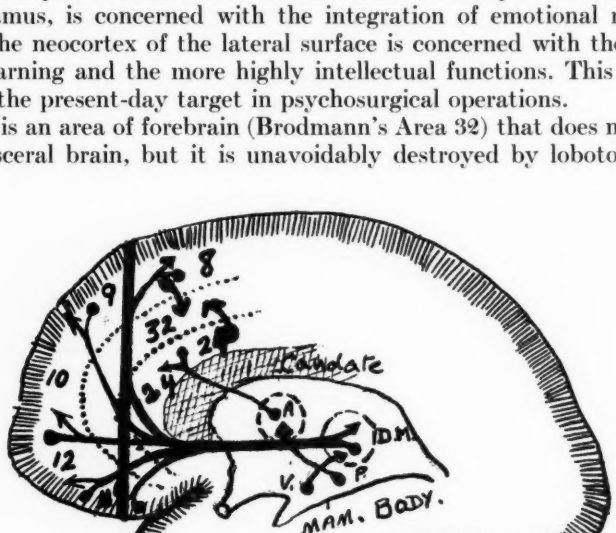

FIG. 1. Major fronto-subcortical pathways (Brodmann's cortical areas, redrawn from Le Gros Clark[2]). D.M. = Dorsomedial nucleus. A. = Anterior nucleus. Mam. Body = Mammillary body. V. = Mammillothalamic tract (bundle of Vicq d'Azyr). P. = Periventricular system of fibres from hypothalamus to dorsomedial nucleus.

mysterious area is subjected to intensive suppressor influences. Meyer and McLardy[10] have referred to it as a nodal point of considerable physiological activity between cortex and subcortical basal ganglia. That it has some concern with cortical activities underlying rather complex psychological processes is suggested by the few observations that have so far been made on the clinical effects of interference with some parts of the hippocampal circuit.

The writer is of the opinion that the destruction of Area 32 is contributory to a successful result. Cingulo-tractotomy passes through Brodmann's Areas 8 or 9, deliberately destroys Area 32 and partly destroys Areas 24 and 11. It also interrupts most of the projections of the visceral brain (Fig. 1).

OPERATIVE TECHNIQUE

Preliminary medication consists of morphine gr. 1/3–1/6 and scopolamine gr. 1/150 administered subcutaneously. Nembutal gr. 1.5 is given orally 1 hour preoperatively. In cooperative patients the whole operation is done under local anesthesia. In other cases pentothal sodium is used.

In the female patient, the frontal half of the head is shaved. In the male, all of the head is shaved. The patient is placed in a semi-Fowler position. The head is prepared and draped in the usual manner. Two parallel incisions, usually 4 cm. in length, in a sagittal plane are made 2.5 cm. from the midline, beginning just in front of the coronal suture and ending behind the hairline. Two mastoid retractors are applied. By means of an electric drill two 1 cm. burr holes are made 3 cm. in front of the coronal suture and 2.5 cm. from the midline. The dura mater is incised in a cruciate manner and its edges are cauterized, thus insuring an almost 1 cm. exposure of the cortex, covered with pia arachnoid and pia mater. The latter two structures together with intervening blood vessels are cauterized and cut. The 1 cm. exposed cortex is cauterized and removed. The incision is deepened until the white matter is seen.

A ventricular needle is introduced and directed towards the edge of the sphenoid wing. A second needle tract is then made more posteriorly at an angle with the first tract at less than 25° down, just missing the anterior horn of the ventricle. Sometimes it has actually entered the ventricle. Between these two tracts lies the desired plane of section. The combined tip is introduced in the first tract. The tip is turned medioposteriorly and with a kind of stroking movements from below upwards the brain tissue is cauterized in a medial and slightly posterior direction until a rubbery feeling is encountered, which means that the falx has been reached. Most of the cauterization is done under vision (using a head light or, better still, a lighted round Sjöqvist retractor). Cauterization is continued mediosuperiorly until the lower border of the sagittal sinus is reached. Then the Gigli guide is introduced and directed medially towards the falx to complete the leucotomy. Complete haemostasis is secured (Fig. 2).

The same procedure is performed on the other side. The burr holes are plugged with two 1 cm. circular pieces of gelfoam. The wound is closed with silk sutures, and dressed with gauze and elastoplast.

The patient is very little disturbed except that the blood pressure falls, for example from 120–90 Hg mm., in from a few minutes to a few hours. It then gradually rises to its preoperative level.

MATERIAL

Cingulo-tractotomy was performed on 28 patients. There were 18 cases of schizophrenia, 1 case of depression, 1 of paranoia, 1 of sexual psychopathy, 1 of intractable pain caused by inoperable intramedullary tumour, and 6 cases of idiopathic epilepsy.

I. *Schizophrenia.* The 18 schizophrenics have been divided into two groups: (a) recent and previously untreated patients, and (b) chronic schizophrenics who have undergone all kinds of treatments, including electric shock therapy.

(a) *Recent* (5 cases). All of these patients were males. Four, whose ages ranged from 18 to 25 years, were operated upon during the first 2 weeks of

their illness. All made complete recovery. The fifth patient, who had had a course of 6 treatments with electric shock without much improvement, was operated upon 1 month after the onset of his illness. He too made a good recovery. He said: "The operation hurt me much less than the electric shock."

All 5 patients went to their homes within 2 weeks, and returned to their work within a month.

(b) *Chronic* (13 patients). There were 4 women and 9 men in this group. The duration of their illness was from 2 to 8 years and their ages ranged

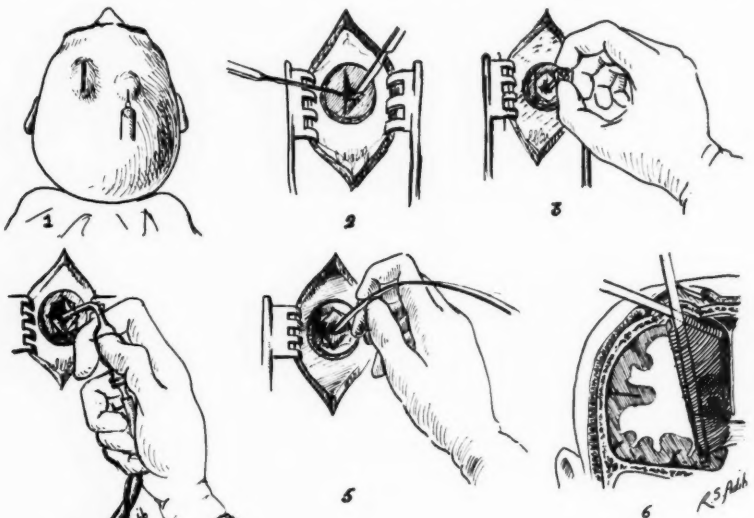

FIG. 2. Diagrams showing technique of cingulo-tractotomy (described in text).

from 19 to 52 years. These patients had had all kinds of treatments previously, including insulin and electric shock therapy. Two of them had also had a previous leucotomy.

Three of the patients (1 female and 2 males) were in excellent condition after the operation. They went back to their homes within 2 weeks, and were at their jobs within a month. However, 2 of these suffered a relapse after 4 months. One was given Cartazol therapy, and although there was some improvement, he did not return to his postoperative status.

Five patients (3 males and 2 females) improved after the operation and all of them were sent home. Their parents and relatives thought they were less aggressive and more manageable than before. They were unable to work although some of them could do knitting, gardening, etc. at home.

Four patients (1 female and 3 males) were not benefited by the operation

They were not made worse. Three of these subsequently had a bilateral prefrontal lobotomy; there was no improvement, and 1 of these patients died from self-induced infection after the lobotomy.

II. *Heterogeneous Group* (depression, paranoia, sexual psychopathy and intractable pain). The 4 cases in this group are presented in the following brief reports.

Depression. The patient, a 42-year-old Mullah, was an intelligent man. He had been very much depressed for the last 6 months. He thought a great deal about Heaven and Hell. He could not sleep and smoked heavily. He was contemplating suicide. He had had all kinds of treatments without much help.

After the operation, he ate well, slept well, and was euphoric. He said, "Doctor, before the operation I was in Hell, now I am in Heaven."

Paranoia. A 60-year-old woman suffered from hallucinations, delusions of persecution, and insomnia. She had become a morphine addict. She complained a great deal from hallucinations of camels and buffaloes rushing into her bedroom. She had various sensations of itching, burning, etc. all over her body. Because of her weakness it was decided to do a unilateral cingulo-tractotomy.

Postoperatively she was free from addiction to drugs and most of her complaints ceased. She can sleep without morphine.

Sexual Psychopathy. A 65-year-old man had become sexually perverted and bestial. He would not have sexual intercourse with his wife, but would try with cows and other animals. During operation gross organic changes and atrophy were evident in his brain.

After the operation he was apathetic for a month. Lately it has been reported that he has reverted to his old habits.

Intractable Pain. A 56-year-old diabetic suffered from an inoperable intramedullary tumour at T9-L1. He complained constantly of severe pains. He had an automatic bladder and was constipated. His mind had become unstable. He had attempted suicide twice.

After the operation, not only his pain but his spasms (mass reflexes) disappeared for 2 months. The spasms gradually began to reappear. He used to describe his experiences with pain without affect, just as though it belonged to someone else. His mental status is stable and he has not attempted suicide. The disappearance of mass reflexes after the operation is interesting. The writer has no explanation for this phenomenon. Their reappearance is a source of worry for the patient and his relatives.

III. *Idiopathic Epilepsy.* The 6 cases in this group will be reported in detail at a later date after a longer follow-up study. Now, 1 year after the operation, it may be said that the results are encouraging.

RESULTS

Of the 18 schizophrenics, 5 were operated upon soon after the onset of their illness. Four, who had had no previous treatment, were operated upon within 2 weeks, and 1, who had had recent electric shock therapy, was operated upon within a month. All 5 made a good recovery and returned to their work.

The 13 remaining schizophrenics had previously been subjected to many

types of treatment, including electric shock therapy. Only 1 of these patients made a permanent recovery after cingulo-tractotomy. Two were well for 4 months and then suffered a relapse. Bilateral prefrontal lobotomy was subsequently performed on 3 patients in this group without improvement.

Of the 4 patients in the heterogeneous group all were improved with the exception of the sexual psychopath.

COMPLICATIONS

Haemorrhage and infection may be prevented by means of careful haemostasis and the administration of Terramycin respectively. There was no postoperative haemorrhage. There was 1 case of self-induced infection (the only mortality in this series).

Vomiting. One third of the patients vomited on the 2nd or 3rd day.

Wetting. About 90 per cent of the patients had this complication from 3 to 7 days postoperatively.

Epilepsy. So far this complication has been nonexistent in this series. The writer believes that its absence may be ascribed to the selective and conservative nature of the operation.

DISCUSSION

It is a contemporary neuropsychiatric truism to apply psychosurgery to chronic and hope'ess cases. Neuropsychiatrists have been mainly responsible for developing this attitude. The neurosurgeons are asked to operate, and are too willing to oblige, still forgetful of von Bergmann's advice, which was also stressed by Horsley and reiterated by Cushing[9]—a neurosurgeon must be his own neurologist. [May I add—and familiarize himself with neuropsychiatry. For is not every operation an experiment in neuropsychiatry and neurophysiology?]

Although "team work" and "group practice" are essential in modern therapy, let no neurosurgeon forget that more important than cooperation with his colleagues is his own independent judgment. Furthermore in the last decade neurosurgeons have concentrated on improving psychosurgical "hows" and neglecting its "whens."

Now having made psychosurgery comparatively safe for the patient, let us make the patient "hopeful" for psychosurgery. In my opinion this "hopefulness" depends above all on submitting the patient to surgery *early* in the course of his illness. I am not unaware of the objections to this opinion that are held by those in the field of neuropsychiatry. I am only presenting a viewpoint and supporting it with some experiments, with the hope that others will repeat these experiments and prove or disprove my viewpoint.

SUMMARY

1. A comparatively simple, safe, selective and effective operation has been described.

2. This operation was performed on psychotic patients.

3. The desirability of early operations, as a first and not the last resort, especially in cases of schizophrenia in which there has been no previous treatment, is emphasized. Five schizophrenics who were operated upon soon after the onset of their illness recovered completely. Of the 13 chronic schizophrenics, only 1 made a permanent recovery.

4. In the heterogeneous group of 4 patients, all made a good recovery except the sexual psychopath.

I wish to express my gratitude to Dr. Salman Faik for referring some of these patients to me, to Dr. Rafid S. Adib for his valuable assistance, and to Sister Asli Malik for her interest in, and devotion to these patients.

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THE SURGICAL TREATMENT OF DYSTONIA MUSCULORUM DEFORMANS*

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THE severe disability of patients afflicted with dystonia musculorum deformans arouses the urge to give them relief even at the cost of other neurologic sacrifices. Unfortunately, the etiology of the disorder is too poorly understood at present to permit a specific attack upon brain areas responsible for the abnormal motion.

The bizarre, yet stereotyped movements do not characterize a disease, but constitute a syndrome for which no specific pathologic lesion is known. The syndrome is considered to operate primarily via the extrapyramidal system. The abnormal involuntary motor activity is regarded as a release phenomenon, the result of loss of the inhibitory action of higher controlling centers. The motions persist too long to be reasonably attributed to an irritative lesion.

Dystonia consists of slow, long-sustained turning movements of the head and trunk, with rotation of especially the proximal parts of the extremities. In addition to these torsion movements, there are slow, sustained tensions in the platysma, shoulder muscles, pectorals and leg and foot muscles. Particularly long-sustained, simultaneous tensions of opposing muscle groups characterize dystonic movements. Physiologically, they resemble those of athetosis, but are longer sustained.

This report concerns the results obtained by surgical interruptions of pathways at several levels in the central nervous system for relief of severe dystonia in 3 children.

CASE REPORTS

Case 1. D.Y. was first seen at the age of 7 years. For a year, she had suffered from spasticity of both arms and from cramping spasms of the feet causing pain that made her cry at night. Within 6 months dystonic movements became pronounced.

Over the next 5 years the dystonia progressed. The head retracted, and the extremities were maintained in semiflexion and were relatively fixed. The facial muscles were not involved, but those of the trunk and extremities forced the body into grotesque postures. These movements were extremely painful and the child was reduced to an emaciated state. Extensive drug therapy was ineffective and she was admitted to the Children's Hospital at the age of 12 years to permit attempts at surgical relief, especially of her pain.

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Operations. Encouraged by the results obtained in cases of unilateral Parkinsonism, on *July 11, 1946* we first partly severed the anterior limb of the right internal capsule by the method of Meyers[2] and Browder.[1] This did not alter the patient's condition.

On *July 22, 1946*, the right area 6 was removed, sparing area 4. This abolished the dystonia and permitted weak voluntary motion of the hand. The right-sided dystonia increased, especially in the foot.

Fearing the effect of a bilateral cortical resection, since the child was intelligent, we sought control of the right side in an attack at the cord level. On *July 30, 1946* the right anterior cord fasciculus was cauterized (Putnam's method[4]) at C1–2 level. This left the child comfortable. The arms and legs were maintained flexed. Right grip was possible and the arm could be extended without eliciting dystonia. Involuntary movement was present in the right leg. Passive extension of the left arm resulted in a strong, coarse tremor and in flexion of the extremities. The neck still retracted to the left moderately under tension or emotion. A more complete denervation of the left side seemed desirable.

On *Aug. 28, 1946* the right area 4 was excised above the level of lip response to stimulation. This resulted in a left hemiplegia, without tremor or reflex spasm.

In the next 9 months all gains were preserved; the child gained 20 pounds, was pain-free, friendly and comfortable in a wheelchair existence. Involuntary movement followed use of the right leg, which was flexed at knee and thigh. In an attempt to relieve this, the right pyramidal tract was severed[5] at the 3rd dorsal level on *May 12, 1947.*

Course. Two years after the last operation (May, 1949) the 15-year-old girl was happy and pain-free. She was 50 pounds heavier than at the start of her surgical "career." Her I.Q. still measured 110 and she was a freshman in the Crippled Children's School. She subsequently has been graduated. Her head was tilted slightly to the right. The spine was in marked scoliosis. She could stand in braces, but could not walk. No dystonia nor resting tremor was present. Right grip was satisfactory and she could write legibly but slowly.

Within the last 3 years, coarse tremor and some dystonia have appeared in the right arm and have progressed until the extremity is almost useless. A left pedunculotomy is being considered to improve this.

Comment. This child has been observed over a period of 10 years. Primarily to relieve pain, successive surgical sacrifices were made to include finally: The right cortical areas 4 and 6, the right extrapyramidal tracts (anterior cord fasciculus) at C1–2 and the right pyramidal tract at D3 level. These resulted in abolition of the dystonia at the cost of a left hemiplegia and a right hemiparesis. The end result was happily accepted by the patient and her relatives.

Case 2. J.N. was a 9.5-year-old boy, admitted to the Children's Hospital on April 25, 1949. The patient was one of twins, the sister being normal. He weighed 3 pounds (1.4 kg.) at birth and lived in an incubator for 3 months. During infancy and after, he had difficulty in eating, eye movements were uncoordinated and his extremities displayed involuntary movements. Despite 4 years of intensive cerebral palsy management his condition deteriorated. For 8 months he had suffered frequent painful attacks of opisthotonos and flexion of the knees and hips.

Examination. He was poorly developed and nourished and weighed 34.5 pounds (15.7 kg.). He lay in bed in severe opisthotonos with hips and knees flexed and arms extended. He could say a few simple words, but speech was greatly inhibited by severe dystonic movements which involved vocalization. Relaxation was impossible. The child was very uncomfortable and sweated profusely.

An encephalogram showed normal ventricles and subarachnoid markings. The spinal fluid was normal.

Operations. On *May 17, 1949*, an attempt was made to remove the right area 4, but because of inconclusive results on stimulation, the precentral gyrus was removed. The specimen measured 1 cm. in width and 1.5 cm. in depth.

Dystonia was abolished on the left side, and persisted on the right; opisthotonos persisted. The left extremities remained in plastic spasticity, with resistance chiefly to flexion.

On *June 1, 1949*, an attempt was made to excise the left area 4, but again this was thwarted by inconclusive results on stimulation. The precentral gyrus was excised in a cerebral sacrifice 4 cm. in diameter.

Thereafter no dystonia was noticed, opisthotonos did not recur and the child was comfortable. He had a spastic quadriplegia with the right arm flexed and the left extended, each resisting contrary passive motion. The legs were extended and crossed.

Course. These gains were maintained at examination 2 years later, except that the legs had become flexed. The child remained severely retarded mentally. He was well-nourished and comfortable. He had developed the ability to say a few words and could move the head and the extremities to a small extent.

Comment. This mentally retarded child's dystonia involved the face, jaws and neck as well as the back and extremities. Removal of areas 4 and 6 (more extensively on the left) controlled the dystonia at the price of spastic quadriplegia and aphasia. The exchange was considered worthwhile by all involved in the care of the child.

Case 3. J. W., a 12-year-old white girl, had been born spontaneously after 7 months of gestation terminated by toxemia. The mother had experienced 7 idiopathic miscarriages.

The child was never able to hold up her head, sit, walk nor use her extremities. At the age of 4 she began having "torsion spasm." Finally she was admitted because intermittent head retraction interfered with breathing, making suffocation appear imminent.

Examination. She was poorly nourished and developed. She lay in opisthotonos with the head retracted and turned to the right. Dystonic movements periodically increased the retraction, throwing into relief the hypertrophied sternomastoids over which coursed the distended jugular veins. As the movement was prolonged the face and chest became cyanotic. The child could be lifted from the floor by a hand under the occiput. Both arms usually were extended and the hands clinched. The left leg was extended and the right flexed. The left hip joint was dislocated and the ankle joint was flail-like. Dystonic and athetoid motion involved all extremities and was sharply increased by sensory stimuli and passive motion. The child apparently understood some words and spoke a very few with great effort. When relaxed she could move all extremities feebly and imperfectly.

An encephalogram revealed essentially normal ventricles and subarachnoid spaces.

Operations. On *Nov. 12, 1951,* the right cerebral peduncle was exposed and the anterior one-third of its lateral surface was incised to a depth of 5 mm. On *Nov. 19, 1951,* the left peduncle was incised similarly through the anterior one-half of its lateral surface.

Course. Postoperatively, the dystonia was abolished. The child could lie comfortably on her back; the neck was supple. The arms were maintained flexed, with the right wrist also flexed. Both legs were flexed, but could be extended within the limitation of her joint contractures. Both legs and arms resisted extension. The right arm also resisted flexion. The child spoke about as much as, but more easily than, before operation. She obviously was markedly deficient in mentality, as she had been before.

Comment. This child's dystonia involved all four extremities and the muscles of the back. Opisthotonos had progressed to a degree threatening suffocation by distortion of the airway. The dystonia and opisthotonos were abolished by interruption of the anterior one-half of the cerebral peduncles. While spastic quadriparesis resulted, the degree of original spasticity was reduced to the point of permitting return of some voluntary motion.

DISCUSSION

Data obtained from these 3 cases suggest that dystonic motion is mediated over both pyramidal and parapyramidal pathways (Fig. 1). It may be abolished at the cortical level by removal of areas 4 and/or 6, above the basal ganglia, by interruption of these pathways in the ventral halves of the cerebral peduncles or in the spinal cord.

In Case 1, gross removal of area 6, sparing area 4 (and, no doubt, a significant amount of adjacent area 6), resulted in abolition of dystonic motion in the contralateral side. Sufficient reflex spasm (i.e., tonic muscular contraction resulting from the application of a stretch-stimulus to a muscle by extending a joint) persisted to be disabling. This was abolished by the subsequent removal of area 4. Dystonia was abolished in the appropriate extremities in Case 2 by gross removal of the precentral gyrus alone.

Similar results followed interruption of these separate pathways in the spinal cord. Unilateral anterior column section abolished the dystonic movement in the leg, but the reflex spasm and some athetosis persisted. These were reduced markedly after pyramidal cordotomy. Unfortunately, the beneficial effects of cord sections are not permanent. Perhaps the impulses subserving the abnormal motion become re-routed over pyramidal and extrapyramidal pathways that escape section in the limited incisions that are possible in human subjects.

Bilateral partial cerebral pedunculotomy is the operation of choice of the three procedures. The results are more favorable and the side-effects are less disabling than are those following cortical operations. The favorable effects include more body area than do those of cord section.

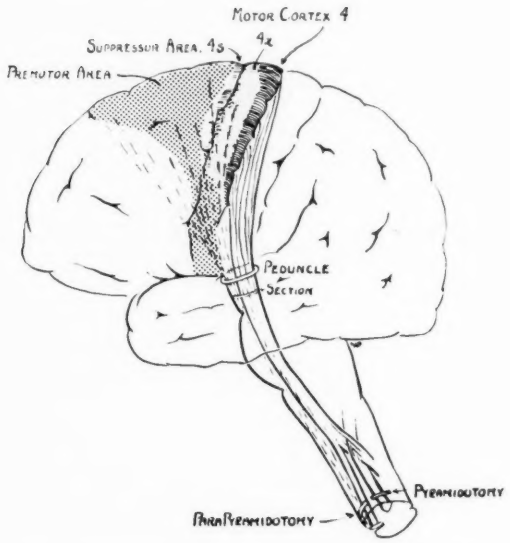

Fig. 1. Diagrammatic representation of the course of the pyramidal and parapyramidal tracts, exclusive of the brain-stem connection.

Case 1. a) Excision right area 6. Abolished left dystonia. Tremor and reflex spasm persisted. b) Excision right area 4 and 6. Tremor and reflex spasm abolished. c) Right parapyramidotomy. Abolished right dystonia. Tremor and reflex spasm persisted. d) Right pyramidotomy. Tremor and reflex spasm abolished.

Case 2. a) Partial excision right area 4 and 6. Abolished left dystonia. Plastic spasticity persisted. b) Partial excision left area 4 and 6. Abolished right dystonia. Plastic spasticity persisted.

Case 3. a) Right pedunculotomy. Abolished left dystonia. Plastic spasticity persisted. b) Left pedunculotomy. Abolished right dystonia. Plastic spasticity persisted.

These experiences are marked by too many variables to assist much in a better understanding of the mechanism of dystonia. The area 4 and 6 decortications were too gross to permit assay of the effects of precise lesions. The peduncle incisions are relative unknowns concerning specific tracts severed and the amounts included. As Meyers[3] has noted, the concepts of anatomists vary widely as to the area and extent in the cross section of the peduncles of the several traversing fiber tracts. It is now appreciated that cross sectional area-maps of the spinal cord fiber tracts are only gross approximations, representing maximal concentrations, with great intermixtures. There is little reason to doubt that a similar state exists in the cerebral peduncles.

It is interesting to speculate upon the possible effects of making sections above and below the points of maximal intake of fibers connecting with the feed-back circuits of the reticular formation, the cerebellum and the basal ganglia. The present experiments show little difference in the motor effects of lesions made above and below the level of the basal ganglia. The results of interruption of fibers between the feed-back mechanisms and the motor cortex still remain to be demonstrated.

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ADDENDUM

Since submission of this manuscript, bilateral cerebral pedunculotomy has been performed on 2 additional children (L.S. and I.S.) for severe dystonia. The early results in these cases resemble those of J.W. (Case 3). The gains reported in the latter case have persisted to date, approximately 22 months.

WOUNDS OF DURAL SINUSES*

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Je le pansai, Dieu le guarit.

AMBROÏSE PARÉ

HARVEY CUSHING's study of war wounds of the brain[2] established the principle of early surgical intervention. His observations during the first World War advanced the idea that operation might serve to prevent the serious sequelae of progressive sinus thrombosis in wounds involving the major venous channels within the cranial cavity. Gordon Holmes' and Percy Sargent's[5] classical study of 70 cases in which the longitudinal sinus was injured, or the circulation disturbed in its immediate neighborhood, forms the very basis of our understanding of the pathology that may be caused by the sudden impairment of intracranial venous circulation. Barnes Woodhall's investigations[10] have shed much light on the anatomy of cranial blood sinuses. On the basis of his experiences during the second World War, Donald Matson[6] has summed up the principles of modern surgical management of dural sinus wounds to which most of us adhere.

It is the purpose of this communication to discuss the surgical management and the neurological sequelae of 112 consecutive wounds of the brain involving major dural sinuses. The surgical treatment of these wounds might serve to suggest a feasible pattern to follow. Our own errors, we should like to think, might help others to improve the record in the future. The analysis of neurological sequelae will be inadequate at best, for in the vast majority of our cases the disturbances of venous circulation were associated with gross cerebral lesions.

SELECTION OF CASES

During a 23-month period from September 1950 through August 1952, 112 consecutive casualties with penetrating wounds of the brain involving dural sinuses were treated at the Neurosurgical Detachments of the Eighth United States Army in Korea (EUSAK) and at the Neurosurgical Center of Tokyo Army Hospital, Japan Logistical Command (JLC).‡ They were

* Presented at the annual meeting of the Harvey Cushing Society, Hollywood, Florida, April 24, 1953.

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‡ The following officers were associated with the author in the care of the casualties on which this report is based: Samuel W. Atkins, Joseph C. Barnett, Edward J. Bishop, Robert A. Clark (USNR), Clifford P. Goplerud, Griffith R. Harsh, III, George J. Hayes, Kim, Sun Keoun (ROKA), Milam Leavens, Lee, Ki Sup (KRC), Henry G. Liss (USNR), John B. Moyar, Jose Ninecurt, Richard R. Patterson, Paul R. Rosenbluth, Gordon T. Wannamaker, and Yoon, Bog Young (ROKA).

United States soldiers and marines, members of the United Nation Forces and of the Republic of Korea Army (ROKA).

The study is limited to cases of trauma of the dural sinuses and does not include in the statistics wounds affecting their tributaries. The following criteria were used to select cases for inclusion in this report: 1. Transection of a sinus. 2. Laceration of a sinus wall perforating the entire thickness of the wall. 3. Contusion and compression of the sinus, in all but 4 cases manifested by abrasions of the outer wall with multiple bleeding points. 4. Thrombosis of an anatomically intact sinus evidenced by surgical inspection and digital palpation.

ANALYSIS

Of 112 consecutive operative cases, 7 were of multiple dural sinus injuries, resulting in a total of 124 individual sinus wounds. There were 13 deaths, a mortality of 11.6 per cent (Table 1).

TABLE 1

Wounds of dural sinuses, September 1950–August 1952, U. S. Army, Korea

Number of consecutive cases	112
Fatal cases	13
Mortality	11.6%
Cases of multiple sinus wounds	7
Number of sinus wounds	124

The pentrating wounds of the brain were caused by shell fragments in 65 cases and by high velocity missiles in 32 cases. Two patients were struck by a rock and 2 by a blunt object. Four patients were involved in a vehicle accident. In 7 instances the mode of injury remained unknown. Thirty of the 112 patients had associated wounds of the body.

There were 78 patients with a wound of the superior longitudinal sinus. The transverse sinus was involved 29 times, but 2 patients had an injury of both the right and left transverse sinus. There were 7 wounds of the torcular Herophili. The sigmoid sinus was involved in 5, and the inferior longitudinal and superior petrosal sinus each in 2 cases. One wound of the straight sinus was encountered (Fig. 1 and Table 2).

Transection of the sinus occurred in 9 cases, in 6 of which the sinus was found to be thrombosed. Laceration of the wall of the sinus, of varying degree, occurred 87 times; a thrombus was encountered at time of surgery at the site of laceration 14 times. Twenty-two cases were classified as contusion and compression; in 19 of these the contusion was manifested by abrasions of the outer wall of the sinus with multiple bleeding points. A thrombus was diagnosed on the basis of surgical inspection in 1 of the 22 cases. Thrombosis of an intact sinus was encountered in 6 penetrating wounds of the brain (Table 3).

Surgically significant intracranial hematomas (Table 4) occurred in 28 of the 112 cases (25 per cent).

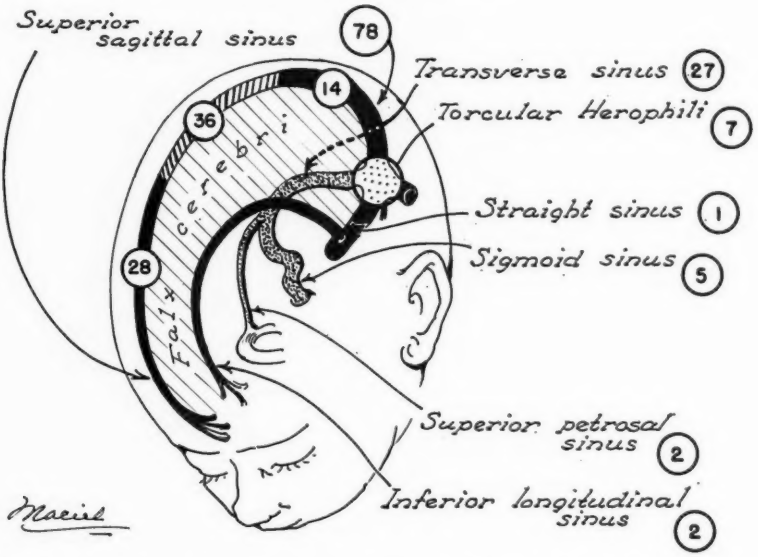

FIG. 1. Wounds of dural sinuses, U. S. Army, Korea, showing number of cases from September 1950 to August 1952.

TABLE 2

Wounds of dural sinuses, September 1950–August 1952, U. S. Army, Korea

Site	No. of Cases	Fatal Cases	Mortality
Superior longitudinal sinus	78	10	12.8%
Transverse sinus	27 (29 wounds)*	3	11.1%
Torcular Herophili	7	0	0
Sigmoid sinus	5	1†	20.0%
Inferior longitudinal sinus	2	0	0
Superior petrosal sinus	2	0	0
Straight sinus	1	0	0

* Two cases of involvement of right and left transverse sinus.
† Also listed as fatal case under "Transverse sinus."

TABLE 3

Type of lesion in 124 dural sinus wounds

Lesion	No. of Cases	Associated Thrombosis
Transection	9	6
Laceration	87	14
Contusion	22	1
Thrombosis	6	—

TABLE 4

Hematomas in 112 cases of wounds of the dural sinuses

Subdural hematoma, homolateral	10
Subdural hematoma, bilateral	2
Extradural hematoma, homolateral	5
Intracerebral hematoma	11
Total	28 (25%)

I. WOUNDS OF SUPERIOR LONGITUDINAL SINUS

(78 cases, 10 fatalities, 12.8 per cent mortality)

Of the 78 patients in this group, 3 had trauma of adjacent sinuses. There were 17 transventricular wounds. In 7 cases the injury involved the frontal air sinuses.

In 6 patients the injury resulted in transection, while the sinus was found to be lacerated in 50. In 17 cases the lesions were classified as contusion and compression, and in 5 instances thrombosis in the presence of an intact sinus wall was diagnosed on the basis of surgical inspection.

Meningocerebral infection was a complicating factor in 11 patients, 3 of whom succumbed to overwhelming cerebritis. Eight patients reached an Army neurosurgical installation from 5 to 7 days after having been wounded and had advanced meningocerebral infection at time of neurosurgical admission. In 3 patients meningocerebral infection developed despite early neurosurgical intervention. (In 1 of these the infection could be clearly traced to an error in surgical judgment on the part of the author.) There were 5 instances of scalp infection.

1. ANTERIOR PORTION

(28 cases, 3 fatalities)

This group includes all wounds anterior to the Rolandic outflow. There were 3 transections and 17 lacerations of the sinus. Thrombosis was encountered in all 3 instances of transection and in 1 case of laceration; it was also present at time of secondary craniotomy in a sinus that had been ligated apparently because of an extensive laceration.

Surgical repair was accomplished by ligation in 2 instances. Eleven lacerations of the sinus were repaired with silk sutures tied over gelfoam or fibrin foam, and 1 was repaired with silk sutures only. Surgical removal of a thrombus was not attempted in any case.

As far as can be determined, all patients were rendered unconscious initially. Except for 3 of them, all were irresponsive to a varying degree at time of admission to a neurosurgical installation. In 6 cases there was no detectable neurological deficit and in 4 cases the deficit was fairly insignificant.

Varying degrees of hyperactivity of deep tendon reflexes with diminished or absent abdominal reflexes, suggestive of unilateral frontal lobe pathology,

were noted in 4 patients. Hemiparesis or hemiplegia, accompanied by the usual reflex changes, was found in 8 patients, but only 4 of these had fully developed spastic hemiplegia. One of the 3 patients who died had a spastic paraparesis.

In 1 instance a large subdural hematoma was encountered despite normal neurological findings. In 4 other cases the neurological deficit could be traced to the presence of a homolateral extradural, a homolateral and a bilateral subdural, and a subcortical hematoma. None of these patients retained a neurological deficit postoperatively.

Representative Case

Case 1. K-28. 23-year-old U.S. Army Private. Penetrating, left frontal shell wound caused by a trip flare, Feb. 9, 1952, involving left frontal air sinus, simultaneously destroying left eye.

Drowsy on admission 2½ hrs. later. Avulsed scalp wound from upper lid of left eye to helix of left ear. Left frontal air sinus and macerated portion of left frontal lobe exposed in area measuring 8×8 cm. Entire wound grossly contaminated with hair and debris. X-ray: Extensive comminution and depression of left frontal bone, left frontal air sinus and left orbit. B.P. 110/70. Pulse 82. Respiration 24. No neurological deficit except for destruction of left optic nerve.

Immediate craniectomy under endotracheal anesthesia with pentothal induction. Upon resection of macerated portion of left frontal lobe, a large bone fragment was found to have lacerated the wall of the anterior portion of the superior longitudinal sinus. Repair was accomplished with interrupted fine silk sutures tied over gelfoam. Primary closure with fascia lata as dural substitute and layer closure of scalp.

Uneventful recovery. Ambulatory on admission to neurosurgical center in Japan. Neurological deficit: partial right 7th nerve palsy of central type.

Fatal Cases. In 2 of the 3 fatal cases the patients were decerebrate on admission. One of them (K-56) died 24 hours after operation. Surgical inspection revealed thrombosis of a transected sinus in the presence of extensive bifrontal damage. The course of the other (K-38) was complicated by a postoperative hemorrhage, removal of which did not alter his neurological status. He never responded after admission, intermittent bouts of extensor rigidity and hyperthermia developed, and he expired 8 days after having been wounded. The third patient (K-57) was not moribund on admission. Bifrontal craniectomy revealed a subdural hematoma overlying the right frontal lobe. Bloody and liquefied brain tissue was removed from the right lateral ventricle. There were extensive destruction of the left frontal lobe. an intracerebral hematoma, laceration of the anterior horn of the left lateral ventricle and tear of anterior portion of the superior longitudinal sinus. The sinus hemorrhage was controlled with silk sutures tied over gelfoam. The patient did well for 24 hours after operation, when signs suggestive of a progressive sinus thrombosis developed, an impression that was confirmed by the appearance of the brain at secondary exploration.

Discussion. The neurological deficit seen with wounds of the anterior portion of the superior longitudinal sinus does not follow any specific pat-

tern. In all but the 3 fatal cases that deficit could have been the sequel of any penetrating wound of the frontal lobe. The exception to this is best illustrated by 2 of the fatal cases (K-56, K-57). The neurological developments in these 2 cases are comparable to those seen with thrombosis of the midportion of the sinus, presenting the picture that Holmes and Sargent[5] have described as the "superior longitudinal sinus syndrome." We presume that in each of these 2 cases (and possibly also in the 3rd fatal case, K-38) the thrombus extended far posteriorly. None of the other 25 cases in this group presented similar neurological changes.

In 2 of the fatal cases (K-38, K-57), we believe, a thrombosis developed subsequent to repair of a bleeding sinus tear with sutures tied over gelfoam.

2. MIDDLE PORTION

(36 cases, 6 fatalities)

This group includes all wounds within the province of the Rolandic outflow. Among them were 3 transections and 21 lacerations. Thrombosis was encountered in the presence of 2 transections and of 6 lacerations.

The sinus was ligated in 1 instance. Twelve lacerations were repaired with sutures tied over gelfoam or fibrin foam. In 2 of these the thrombus was removed prior to repair. One laceration was closed with silk sutures and 1 small tear was sealed with silver clips. A muscle stamp was used in 3 instances.

Nine patients were oriented at time of neurosurgical admission. Among them was 1 with a left parietal missile wound from which brain tissue exuded and who had a spastic triplegia. Ten patients showed varying impairment of responsiveness. Twelve were admitted in semicoma or coma. Records of 5 others did not contain adequate information.

There was no detectable neurological deficit before and after surgery in 3 patients, and in 2 others the deficit was limited to fairly insignificant changes. One patient had a monoplegia. Seven were admitted with hemiplegia or hemiparesis which was associated with a varying degree of hypertonicity. There were 4 instances of paraplegia or paraparesis with spasticity present in 1 of them. Spastic triplegia or triparesis occurred in 13 patients with flaccid paralysis of the arm and spasticity of both legs in 1. Two patients were quadriplegic; 1 showed pronounced flaccidity of all extremities while the other had marked spasticity.

Representative Case

Case 2. K-70. 27-year-old French soldier. Biparietal penetrating shell wound, Mar. 7, 1951.

Stuporous on admission 24 hours later. BP 150/40. Pulse 74. Respiration 30. Macerated, contaminated wound extending diagonally across vertex with liquefied brain tissue exuding. Spastic triparesis, sparing left arm. Withdrawal to pain stimuli of all extremities. Deep tendon reflexes hyperactive. Left abdominal reflexes diminished. Moderate nuchal rigidity.

Immediate biparietal craniectomy revealed extensive depression and comminution of both parietal bones. Comminuted fragments had torn the superior wall of the longitudinal sinus for a distance of 3 cm. Removal of the thrombus, which filled the entire gap, resulted in hemorrhage. Repair of the lacerated sinus was accomplished with interrupted silk sutures tied over gelfoam. Nineteen bone fragments were removed from the missile tract, which was traced to a point 2 cm. caudally to the inferior margin of falx. Resection of liquefied and necrotic brain tissue did not define a precise zone of demarcation. Anticipating the necessity of a second-stage resection, the dural defect was not grafted, but a piece of gelfilm was inserted extradurally and primary closure of the scalp was performed.

Prior to secondary craniotomy 13 days later, the wound edges had separated and necrotic brain tissue presented at the surface. There was a clearly defined zone of demarcation. No retained bone fragments and no residual hematoma were encountered. The missile tract contained xanthochromic fluid. Evacuation of this fluid revealed a tear of the 3rd ventricle which had not been recognized at preceding operation. Fascia lata was used as dural substitute. The scalp was closed in layers. Subsequent scalp infection necessitated frequent saline irrigations and treatment with plasma powder. The scalp defect was allowed to granulate over. Meningitis did not develop.

On admission to neurosurgical center 49 days later, patient was alert and oriented. No aphasia. Spastic triparesis with virtual paralysis of right arm, but only moderate weakness of left leg persisted. No evidence of meningocerebral infection.

Within 3 weeks, patient had two focal seizures and was placed on Dilantin. He had no further seizures until his return to France where he did not receive any Dilantin.*

Follow-up data are based on numerous letters received from the patient and from members of his family. At time of this writing, 2 years after injury, he walks without assistance and has recovered some use of his right arm. His letters are intelligent and well coordinated. His handwriting is neat and legible. He has travelled on several occasions without being accompanied, but he is not employed.

Comment. The neurological picture presented by this patient shortly after having been wounded is essentially that of Gordon Holmes' "superior longitudinal sinus syndrome." The temporary postoperative change from triparesis to virtual triplegia is of interest in view of the fact that the thrombus was removed at time of first operation. The decrease and eventual disappearance of spasticity is in keeping with the classical observations of Holmes and Sargent.

Continued liquefaction subsequent to initial surgery has been observed in many instances of trauma of the midportion of the superior longitudinal sinus and also in some wounds of the posterior portion of the sinus. More often than not, continued liquefaction and delayed demarcation necessitate a second-stage operation. Optimum time for secondary resection may lie between 4 and 7 days. Postponement for 13 days as in the present case was an error in judgment on the part of the author which could have resulted

* The Overseas Division of Parke, Davis and Co. has now donated Dilantin to this soldier in order to permit him continuation of treatment.

in meningocerebral infection and which may be responsible for the slow initial recovery.

Surgical removal of the thrombus as was done in this case may well be indicated whenever technically feasible. Its removal may be an essential factor in the prevention of blood-stream infection. Restoration of the sinus wall subsequent to removal of the thrombus may lead to the re-establishment of adequate venous drainage.

Fatal Cases. In 3 of the 6 fatal cases of wounds of the midportion of the superior longitudinal sinus the patients died of extensive meningocerebral infection. They did not receive definitive neurosurgery until 11 to 17 days after having been wounded (K-4, K-99, K-100). Two patients died within 12 hours after having been operated upon. One (K-43) had dilated and fixed pupils on admission, and a spastic paraplegia. Operative findings included an almost completely thrombosed superior longitudinal sinus. The other (K-45) also had fixed and dilated pupils on admission, and a spastic triplegia. The lacerated sinus was completely obstructed in its midportion by a solid thrombus.

In 1 patient (K-9), who exhibited a spastic triplegia on admission, thrombosis of the sinus with extensive liquefaction of both hemispheres developed subsequent to repair of a large sinus tear with sutures tied over gelfoam. He died 6 days after having been wounded.

Discussion. As anticipated, wounds of the midportion of the superior longitudinal sinus involving the Rolandic outflow are accompanied by a greater mortality and by a much more devastating neurological deficit than any other sinus wounds. Although in the majority of cases in this group, the disturbances of venous circulation were associated with gross cerebral lesions, the high incidence of paraplegia, triplegia and quadriplegia with prevailing spasticity points to the specific syndrome that has been described by Holmes and Sargent.[5] Cerebral edema and actual liquefaction were striking features in these cases. More often than not, such liquefaction may continue for some days after sinus repair, suggesting a two-stage operation as the surgical method of choice in the management of wounds of the midportion of the superior longitudinal sinus.

Our figures relative to the incidence of thrombosis in this entire series may not be correct. In some of the cases, lacunar thrombosis as suggested by Holmes and Sargent[5] may well have been present despite bleeding from the lumen of the sinus. Also inspection and digital palpation of an intact or contused and abraded sinus wall is not entirely reliable.

3. POSTERIOR PORTION

(14 cases, 1 fatality)

This group includes all wounds posterior to the Rolandic outflow. There were 12 lacerations and 2 contusions with bleeding abrasions of the wall of the sinus. Thrombosis was encountered 3 times in the presence of a sinus laceration. Injury extended to adjacent sinuses in the 3 cases.

Surgical repair was accomplished by direct suture of the sinus wall in 1 case and by sutures tied over gelfoam or fibrin foam in 3 instances. Three sizable lacerations were sealed with muscle stamps. Gelfoam without suture was employed 5 times.

Two patients did not experience unconsciousness when wounded; a 3rd man was rendered unconscious momentarily. The remaining 11 patients showed marked impairment of their responses on neurosurgical admission. Four of them were deeply comatose; 6 also exhibited excessive restlessness.

There was no motor weakness in any of these patients. Hyperactive deep tendon reflexes were found in 3 cases with impairment of the abdominal reflexes in 2 of them. In 3 cases in this group there was bilateral impairment of air conduction.

As anticipated, visual disturbances were predominant in this group, but such impairment could invariably be traced to embarrassment or to direct damage of the visual cortex. Subjective blurring of vision was the only disturbance in the presence of one sinus tear caused by an indriven bone fragment. Vision was initially limited to light perception in 3 patients but was restored at time of evacuation, except for persisting homonymous hemianopia in 2 of them. Two men had a homonymous hemianopia on admission and there was no change in the visual fields after operation. Four patients were rendered totally blind. At time of evacuation from the center in Tokyo, 1 of them could count fingers at a distance of 1 ft. and 1 at a distance of 4 ft. The 3rd retained a homonymous hemianopia. Only 1 patient remained blind.

Representative Case

Case 3. K-104. 21-year-old Marine PFC. Penetrating left occipital shell fragment wound with exuding necrotic brain tissue, Dec. 7, 1950. Associated wounds included frost bite of toes and fingers.

Stuporous and exceedingly restless on admission same day. BP 124/60. Pulse 42. Respirations 16. Pupils equal, fixed to light. No motor impairment. Bilaterally hyperactive deep tendon reflexes. Bilateral Babinski response. Absent abdominal reflexes.

Bi-occipital craniectomy revealed extensive destruction of brain tissue and penetration of bone fragments to a depth of 7 cm. Posterior portion of superior longitudinal sinus was covered by a large blood clot which contained many comminuted bone fragments. Continuous ooze of venous blood from this area suggested sinus laceration which came into sight after removal of clot. Two bone fragments partially sealed the large sinus tear. Their removal resulted in frank hemorrhage. The tear in the sinus was sealed with a muscle stamp from the leg. Closure was performed in layers, deferring radical resection for a second-stage operation. This was accomplished 8 days later. A third bone fragment, which had been overlooked at time of first operation, was found to be lodged in the sinus wall anterior to the previously described tear. Its removal resulted again in hemorrhage from the sinus, clearly indicating that preceding muscle repair had not resulted in thrombosis. The old muscle stamp was replaced by another piece of muscle sealing the sinus laceration, which measured

approximately 4 cm. Additional debridement revealed also a laceration of the posterior horn of the right lateral ventricle.

Though there was primary wound healing, the patient had a stormy postoperative course. He was mentally confused for about 1 month but was rational and oriented at time of evacuation 2 months after having been wounded. He remained blind.

In a letter typed by the patient 2 years later he gave an account of his present activities. He is undergoing training for a civil service job as typist on a dictaphone machine. He engages in dancing and swimming, and lives an active life. His only complaint is of occasional mild headaches. To date, he has had no seizures.

Comment. Muscle repair of a large laceration of the sinus did not result in thrombosis as proven by second-stage operation.

Fatal Case. In the only fatal case in this group (K-66) the patient had sustained a close-range gunshot wound resulting in extensive laceration and thrombosis of the sinus. On admission 3 hours after having been wounded he was in profound coma and exhibited intermittent respiratory failure as well as disturbances of temperature regulation. He expired 2 hours postoperatively.

II. WOUNDS OF THE TRANSVERSE SINUS

(27 cases, 3 fatalities)

The transverse sinus was involved unilaterally in 25 and bilaterally in 2 cases. In 7 patients the missile wound was responsible also for damage to adjacent sinuses; 6 of these had involvement of the torcular Herophili. The 7th patient had a separate laceration of the sigmoid sinus and is mentioned as the 1 fatal case in that section. There was 1 transventricular wound in this group.

Meningocerebral infection was a complicating factor in 5 cases; in 2 of them the infection was present at the time of neurosurgical admission.

In 3 cases injury resulted in transection, and in 21 instances in laceration of the transverse sinus. Depressed bone fragments produced contusion and abrasions of the sinus wall with multiple bleeding points in 3 patients. Thrombosis was encountered in 2 transections and in 2 lacerations. One transected sinus was ligated. Two lacerations were repaired with silk sutures, 11 with sutures tied over gelfoam or fibrin foam, and 4 with muscle stamps. Gelfoam pledgets were employed 7 times. Three patients did not require repair because of existing thrombosis, and in 1 the mode of repair is not known.

Because of their topography, missile wounds of the transverse sinus produced varying neurological deficits. Impairment of vision was particularly common. Cerebellar damage occurred in some, and motor and sensory changes in others. In each case there was definite relation between the neurological deficit and the gross cerebral or cerebellar lesions. None of the neurological changes could be ascribed with certainty to the lesion of the transverse sinus itself.

Representative Case

Case 4. K-65. U.S. Army Second Lieutenant with penetrating left occipital shell wound. Momentary loss of consciousness with subsequent complaint of severe headaches and blurring of vision. Debridement of the wound was carried out in an intermediate station, but no details are available until admission to neurosurgical center in Japan 17 days later.

He was then alert and oriented. Neurological deficit was limited to right homonymous hemianopia. X-ray: Perforation of left suboccipital bone. Comminuted bone fragments lodged in plane of tentorium. Metallic foreign body within left occipital lobe.

Left occipital and suboccipital craniectomy revealed an extradural hematoma overlying the cerebellar hemisphere and the left occipital lobe. One bone fragment was lodged in the transverse sinus. Its removal resulted in sinus hemorrhage which was controlled by sutures placed through the torn edges of the sinus wall. Two of these sutures were tied over a pledget of fibrin foam. No subdural hematoma was encountered. Primary closure without drainage.

Except for persistent right homonymous hemianopia, patient made an uneventful recovery.

Comment. The bone fragment was lodged within the sinus wall for 17 days without producing thrombosis of the sinus. This patient is one of very few in whom meningocerebral infection did not develop despite incomplete initial debridement.

Fatal Cases. K-20 was admitted with fungating cerebritis in the presence of a transventricular wound. Operation revealed a 3-cm. tear of transverse sinus, which was thrombosed. Subsequent ventriculostomy did not result in improvement. Autopsy revealed extensive basilar meningitis with obstructive hydrocephalus. The author, who operated on this patient, failed to institute open treatment, a method which has proven beneficial in the management of cerebritis complicating penetrating wounds of the brain.[8]

K-40 had a through-and-through, fungating, left parietal-left suboccipital wound with subdural hematoma in posterior fossa and over left cerebral hemisphere. The transverse sinus was transected and thrombosed. Patient died in respiratory failure 2 days postoperatively.

The 3rd fatal case (K-82) will be discussed in the section on wounds of the sigmoid sinus.

III. WOUNDS OF THE TORCULAR HEROPHILI

(7 cases, 0 fatality)

The torcular Herophili was involved 7 times. Only 1 isolated laceration of the confluens of the sinuses was seen. The other 6 patients had also involvement of one or more adjacent sinuses. In 1 instance, the lacerated torcular was thrombosed. Shell fragments were responsible for 6 cases. One man sustained a through-and-through gunshot wound resulting in comminution and depression of occipital and suboccipital bones. This did not produce a frank laceration of the torcular, but an extensive contusion of the wall

of the confluens with multiple bleeding points on its abraded surface. None of the patients died. Meningocerebral or superficial scalp infection did not complicate recovery.

In 1 instance the torcular was repaired with silk sutures which were tied over gelfoam. Pledgets of gelfoam were used in 4 cases; in 1 of these the pledget was replaced by a muscle stamp in the second-stage operation. Similar muscle stamps were used in 2 other instances.

Only 2 patients showed motor impairment. One had mild weakness of the lower extremities, but regained all function. The other, K-32 (see Case 5 below) was triplegic but recovered.

There were no visual disturbances in 1 case. Two patients had only light perception when first seen; one of them recovered full vision, the other retained a right homonymous hemianopia. One man could recognize objects prior to surgery. At time of evacuation he had fair vision, sufficient to get about the ward. Two, or possibly 3, patients were blind initially; within 2 weeks after the first operation one of them could count fingers at 1-ft. distance. The other was able to count fingers at 4-ft. distance within 3 weeks. Visual fields checked at a visual angle of 3/330 and 6/330 did not reveal a defect at that time. The 3rd patient was too disturbed mentally to permit determination of his visual status.

In all cases the pupillary light reflex was intact when the patients were first seen; reaction to light and in accommodation was intact in all of them at time of evacuation. One patient showed left inferior conjugate deviation and paralysis of upward gaze. The extraocular movements were fully restored at time of evacuation. Nystagmus was seen in 1 instance. Papilledema did not develop in any patients. While all 7 showed various degrees of impairment of the conscious level initially, only 1 patient remained disoriented at time of evacuation.

Representative Case

Case 5. K-32. 19-year-old U.S. Marine Corporal with gaping shell wound extending from inion to midportion of neck.

Irresponsive on admission same day. Restlessness manifested by feeble, purposeless movements of right arm. Intermittent tonic contractions of left arm. Flaccid paralysis of both legs. Dilated pupils reacted to light. Eyes fixed in left lateral conjugate deviation. Upward conjugate movements could not be induced. BP 140/90. Pulse 120, thready, irregular. Respiration 52, shallow excursions. Hemoglobin 9.5 gm. RBC 3,070,000. X-ray: Comminution and depression of occipital and suboccipital bones. No fracture of cervical spine.

Following 24-hour period of oxygen administration and whole blood transfusions, vital signs stabilized except for respiratory rate which remained 52.

Because of likelihood of brain-stem and midbrain compression, and of dural sinus impairment, bi-occipital and bilateral suboccipital craniectomy was carried out as a first-stage procedure. A large bone fragment had torn the torcular Herophili; the tear extended into the superior longitudinal sinus, which was gaping, and into both transverse sinuses. The lacerated portions of the venous sinuses and the torcular

were thrombosed. Repair was accomplished with gelfoam pledgets. Closure was unsatisfactory because of extensive tissue loss in lower portion of wound.

Extraocular movements returned 4 hours after operation. There was some slight return of motor power in the right leg and in both arms within 4 days, during which time the patient began to respond but seemed to be blind. After 1 week he was fully oriented.

Second-stage craniotomy was performed 19 days later. This operation was necessitated also by a cerebrospinal fluid fistula draining clear fluid from the lower portion of the wound. Necrotic cerebellar tissue presented beneath the skin surface. During partial resection of the right cerebellar hemisphere, a fresh hemorrhage occurred from the right transverse sinus tear which was controlled by silk sutures tied over gelfoam. The medial portion of the left cerebellar hemisphere, which was necrotic, also was resected. Gelfoam with which the torcular had been sealed at first operation was removed with resulting bleeding from the confluens of the sinuses. A piece of vastus lateralis was placed into the open torcular. Dural closure was performed with fascia lata.

At time of evacuation to the Zone of Interior, 49 days later, motor weakness of right arm and left leg persisted, not sufficient, however, to interfere with ambulation in a walker. Vision had been regained sufficiently to permit finger counting at 4-ft. distance.

Two years after having been wounded the patient was re-examined by Dr. C. E. Brackett:* "The patient is enjoying excellent health at the present time. He is employed as mimeograph operator. He walks with no limp and has noted only slight clumsiness of the right hand. He is not able to see as well as to be able to read print. However, his vision is serviceable for getting about. He is completely free of headaches, dizziness, or any other complaints referable to the central nervous system.

"Gait normal except for slightly decreased associated movements of right arm. Station normal. Romberg negative. Succession movements slightly diminished in right hand. Slight hesitancy in finger-to-nose test on the right. Otherwise cerebellar testing within normal limits. Reduced sensation to all modalities in left leg below groin. No evidence of muscle weakness. Reflexes slightly hyperactive in arms, normally active in knees, and hyperactive in ankles, but equal bilaterally. No pathological reflexes. Right homonymous hemianopia with macular sparing. Moderate degree of optic atrophy bilaterally. No venous engorgement. Extraocular movements within normal limits. The defect over the torcular is smooth. Some pulsation is noted in the wound which appears to be entirely well healed. Electroencephalogram normal."

Comment. Analysis of the 7 torcular wounds reveals a striking paucity of neurological changes except in K-32 (Case 5) described above. The deficit in that particular case was probably the sequel of existing compression of brain stem and mesencephalon.

Predominant in all but 1 of the cases was the impairment of vision, which could be explained on the basis of direct cortical contusion. The possibility exists, however, that defective venous drainage was responsible for dysfunction of the visual cortex and that subsequent restoration of vision was facilitated by collateral circulation. It might also have been brought about

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by the re-establishment of venous flow after surgical repair of the venous channels. Free flow of venous blood was seen at second operation in 2 cases even though 1 of them revealed a thrombosis of the torcular initially.

It was Dandy's impression[3] that thrombosis of the torcular Herophili may not be compatible with life because of insufficient collateral circulation for both the longitudinal and straight sinuses. This analysis seems to indicate that temporary occlusion of the confluens can be tolerated, an argument in favor of repair of a lacerated torcular after surgical removal of the thrombus.

IV. WOUNDS OF THE SIGMOID SINUS

(5 cases, 1 fatality)

The sigmoid sinus was involved 5 times. There were 4 isolated lacerations. In one case a .45 caliber bullet, after lacerating the sigmoid, transected the transverse sinus and traversed the cerebellar hemisphere. A burp gun was responsible for 1 of the sigmoid sinus lacerations. The remaining 3 were caused by shell fragments. Meningocerebral and superficial scalp infections were not complicating factors in any of the cases.

In 1 instance the laceration was repaired with interrupted silk sutures; in 2 cases a muscle stamp was used. One laceration was repaired with silver clips which were covered with a gelfoam strip. In the 5th case the method of sinus repair is not known.

Because of the topography of the entrance wound, a peripheral facial palsy of varying degree was encountered in 3 out of 5 cases. Nerve deafness was found in 2 instances. Motor paralysis was not seen. In line with the associated cerebellar damage, a considerable cerebellar deficit was encountered in 4 out of 5 cases.

Representative Case

Case 6. K-114. 24-year-old U.S. Army Corporal with penetrating right temporo-occipital shell wound.

Drowsy, but oriented on admission 24 hours later. Headaches, vomiting. BP 110/60. Pulse 96. Respiration 22. Questionable right 7th nerve palsy. No acoustic perception on the right.

X-ray: Depression, comminution of inferior portion of right temporal and occipital bones, and of right mastoid and petrous bones. Innumerable comminuted bone fragments at base of posterior portion of middle fossa. One metallic fragment within comminuted area of petrous bone.

Operation revealed dural tear at base of middle fossa. Liquefied brain tissue, clots and comminuted bone fragments encountered extradurally. Intradurally a substantial clot and 5 comminuted bone fragments were removed together with liquefied brain tissue. The metallic fragment had pierced the wall of the sigmoid sinus, occluding it temporarily. Its removal resulted in profuse hemorrhage. Repair was effected with silver clips and gelfoam pledgets.

Primary healing and uneventful recovery. Re-examination at the center in Japan revealed no neurological deficit, except for complete loss of hearing on the right and a peripheral 7th nerve palsy on that side.

Fatal Case. In K–82 the operative findings included a transected and thrombosed transverse sinus and a separate laceration of the sigmoid sinus. The patient died of postoperative pulmonary complications 7 days after having been wounded. In the 13 fatal cases in this entire series, he was the only patient to succumb to pulmonary pathology.

V. WOUNDS OF THE INFERIOR LONGITUDINAL SINUS

(2 cases, 1 fatality)

One laceration of the inferior longitudinal sinus and 1 thrombosis in the presence of an intact sinus wall were seen.

Representative Case

Case 7. K-53. U.S. Army Corporal sustained a shell wound of the left forehead resulting in a huge gaping laceration, measuring 8 cm. in diameter, and in destruction of the anterior portion of the left frontal lobe.

At operation a hematoma was encountered covering the floor of the left anterior fossa. Its removal revealed laceration of the inferior longitudinal sinus which was repaired with a muscle stamp.

The postoperative neurological deficit was limited to personality changes of frontal lobe type.

VI. WOUNDS OF THE SUPERIOR PETROSAL SINUS

(2 cases, 0 fatality)

Of the 2 cases in which the superior petrosal sinus was involved, 1 is here reported as a representative case.

Case 8. K-117. U.S. Army PFC sustained a right temporo-occipital, through-and-through bullet wound. Debridement was performed at an intermediate station, but details of the extent of the operation are not on record.

On admission to neurosurgical center in Japan, 7 days later, patient was irrational and exceedingly restless. He had a left hemiparesis and mild nuchal rigidity. Examination of the head revealed a sutured linear incision in right posterior temporal region, overlying a bulging and poorly pulsating defect. Cerebrospinal fluid was noted to leak through the approximated skin edges, which were reddened and edematous.

X-ray: Bony defect in right temporo-occipital area. One cluster of bone fragments in temporal area; a second cluster intracerebrally just cephalad to the plane of the tentorium.

Right temporo-occipital craniectomy was performed 2 days following admission after patient had been placed on antibiotic therapy. Upon opening the scalp incision large amounts of necrotic and partially liquefied brain tissue exuded through a tear in the dura mater. The entire necrotic portion of the temporal lobe was resected. A large hematoma was encountered in the vicinity of the fractured petrous bone. Within this hematoma, 3 comminuted pieces of bone were found, all of which had pierced the wall of the superior petrosal sinus. Their removal resulted in frank hemorrhage which was controlled by a piece of muscle. Gelfilm was used in lieu of a dural graft.

In a second-stage operation, 7 days later, the exit wound in the anterior temporal

region was exposed and 19 extradural bone fragments were removed. As posteriorly, gelfilm was used in lieu of a dural graft.

Recovery was slow but steady. Left hemiparesis subsided and the patient was ambulatory at time of evacuation to the States.

VII. WOUNDS OF THE STRAIGHT SINUS

(1 case, 0 fatality)

Following is the only case in this series in which the straight sinus was involved.

Case 9. K-84. The patient sustained a penetrating left occipital missile wound. He did not receive neurosurgical attention until admitted to the center in Japan.

He appeared lethargic and restless. Inspection of the head revealed three separate penetrations of the scalp in the left occipital area near the midline. Necrotic brain tissue and clots exuded spontaneously from all three wounds. The neurological deficit was limited to a right homonymous hemianopia. There was no papilledema.

X-ray: Depression, comminution of both occipital bones, with intracerebral bone fragments in medial aspect of left occipital lobe.

Bi-occipital craniectomy revealed a narrow, longitudinal defect involving both occipital bones. Necrotic brain tissue with a foul odor, clots and comminuted bone fragments exuded through the bony defect after the scalp had been retracted. The dura mater was widely torn. The left occipital lobe was macerated and had to be resected. Granulation tissue and organized clots covered medial aspect of falx and tentorium. Removal of clots resulted in massive hemorrhage from a large tear in the straight sinus. The tear was repaired with a muscle stamp which was covered with strips of fibrin foam.

Because of existing cerebritis, secondary resection, as anticipated, and primary closure were performed without dural graft.

Reopening of the scalp incision 13 days later revealed a well delineated fungating mass which was found to be firmly in place. Fascia lata was used as dural substitute.

Primary closure resulted in uncomplicated recovery. At time of evacuation to the States, 40 days later, the neurological deficit consisted of marked anomia, cortical sensory loss and right homonymous hemianopia.

Comment. Matson[6,7] has reported 1 case of laceration of the straight sinus. His patient sustained a penetrating transventricular missile wound of the left occipital lobe. The missile tract extended below the falx and contained many comminuted bone fragments. A profuse hemorrhage from the straight sinus was controlled with silver clips.

SURGICAL COMMENTS

As Matson has pointed out,[6] sinus injuries can usually be anticipated from the location of the visible wound (Fig. 2) and examination of X-ray films. Such anticipation will help to prevent undue loss of blood and will facilitate in many instances the surgical re-establishment of venous circulation.

Proper positioning on the operating table of patients with dural sinus wounds has been stressed by many writers. Small and Turner[9] advocate a

reclining position. Evans[4] points to the grave danger of fatal air embolism if the head be elevated at time of operation, and prefers lowering of the head despite increased bleeding from such posturing. A tragic complication of this kind was experienced by the author during the Okinawa campaign (not included in the present series). Three separate tears were encountered in the superior longitudinal sinus. The patient suddenly expired on the operating table after two lacerations had been repaired. Autopsy revealed air embolism as cause of death. There can be no doubt that Evans' method provides the greatest degree of safety.

FIG. 2. *K-50.* Through-and-through bullet wound with laceration of midportion of superior longitudinal sinus.

Pentothal sodium induction, employment of an endotracheal tube, and gas has been our anesthesia of choice. The necessity of maintaining a normal blood pressure before, during and after operation scarcely needs emphasis. The use of artificial hypotension in cases of suspected wounds of the dural sinuses is not indicated.

The cardinal principle of management of sinus injuries, not to disturb the point of laceration of the dura mater until the area has been well exposed, has already been stressed by Matson.[6] It is also important to evacuate all clots which may obscure a sinus laceration, and to remove all comminuted bone fragments that may have lodged in the wall or in the lumen of the sinus. This is necessary for the sake of radical debridement in order to prevent infection; it is also necessary in order to permit surgical restoration of the lumen of the sinus. The tendency for rapid re-establishment of venous circulation in a repaired dural sinus has been demonstrated by Sir Hugh Cairns and his associates.[1]

The removal of a thrombus from the transected ends of a sinus is indicated only as another measure to prevent infection. It permits debridement of the sinus wall which will have to be followed by ligation. To our knowledge, no satisfactory method of repair of a transected sinus has been reported.

Primary thrombosis of a lacerated sinus, which was encountered 14 times in this series, presents an interference with venous circulation that can be eliminated. In 2 instances (K-70, K-77), a thrombus which fully occluded the lumen at site of laceration was removed. The degree of functional return in both these cases seemed to indicate the establishment of adequate venous drainage. We believe that the surgical removal of an obstructing thrombus from a lacerated sinus is warranted.

TABLE 5

Surgical repair of 96 transected or lacerated sinuses

Method	No. of Cases
Ligation	5
Suture	6
Suture tied over gelfoam or fibrin foam	42
Muscle stamp	15
Gelfoam pledget	15
Thrombus removed prior to repair	3
Not repaired because of thrombosis	8
Unknown	5

Methods of surgical repair of the 96 transected or lacerated sinuses in this series have been compiled in Table 5. A suitable technic for the repair of small or moderate-sized lacerations is the use of interrupted silk sutures on eye needles, placed through the edges of the tear. The laceration of the sinus wall can be firmly occluded by tying these sutures over a strip of gelfoam (Fig. 3).

A muscle stamp is the method of choice for the repair of extensive lacerations. As has been shown by Cairns,[1] the establishment of adequate venous drainage in cases similar to K-104 (Case 3) suggests that a large muscle stamp does not necessarily interrupt the flow of blood through a sinus so repaired. The muscle stamp should be about twice the diameter of the laceration. This permits insertion of a small portion of the stamp through the laceration without occluding the lumen of the sinus. The external portion of the muscle stamp prevents oozing and has a cushioning effect on the sinus wall surrounding the laceration. Occasionally it may be necessary to secure the stamp to the dura mater with a few interrupted silk sutures, or to seal it peripherally with a strip of gelfoam.

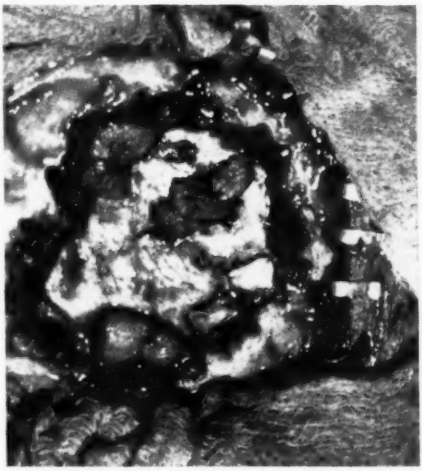

FIG. 3. *K-50.* Laceration of superior longitudinal sinus repaired with silk sutures tied over gelfoam.

In 3 of 42 lacerations, repaired with silk sutures which were tied over gelfoam or fibrin foam, secondary thrombosis developed. The same was observed in 1 out of 15 lacerations that were repaired with a muscle stamp. Thrombosis may, of course, have developed in other instances without manifesting itself clinically. It seems that the occurrence of secondary throm-

bosis depends not so much on the method of repair as on the maintenance of blood volume and pressure before, during and immediately after operation.

SUMMARY

1. A group of 112 consecutive cases of dural sinus wounds, incurred in the Korean War during the period September 1950 to August 1952, has been analyzed. Their neurological sequelae have been described.

2. The surgical management of dural sinus wounds has been discussed.

The author is indebted to Dr. Joseph P. Evans, who has made the preparation of this study possible.

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EXPERIMENTAL STUDIES ON THE CIRCULATION OF THE CEREBROSPINAL FLUID

AND METHODS OF PRODUCING COMMUNICATING HYDROCEPHALUS IN THE DOG*

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MUCH of the experimental work that has been done on the physiology of the cerebrospinal fluid has been performed on laboratory animals and the results have then been applied to man. The purpose of the present work was to discover any differences that might exist among the cerebrospinal fluid (CSF) circulations of the dog, the cat, and the Rhesus monkey, so that the comparisons that have been made between these circulations and that of man might be evaluated. A further objective was to find a satisfactory preparation for the study of communicating hydrocephalus in animals.

Our concept of the circulation of the CSF is essentially that which was described by Weed,[4] as far as the flow and absorption are concerned. In our experiments we have not been concerned with the origin of the fluid, or with following the course of the ionized constituents, about which there is controversy and in which differences may be found to exist between various species and man. Our studies have been to determine the main pathways of flow and absorption beyond the ventricular system in the animals under consideration. At this stage of the circulation the fluid passes into the extracerebral subarachnoid spaces, where it is partly absorbed by arachnoidal villi situated not only along the superior sagittal sinus but also in conjunction with the cavernous and other sinuses. An additional route of absorption exists along the arachnoidal sheaths of certain cranial nerves, from which the fluid passes into intercellular tissue spaces and then into the lymphatic system. For the purposes of this study any fluid and ion exchange which may take place between the CSF and brain substance has been disregarded.

CSF Absorption in the Dog. The existence of pathways along the cranial nerves of dogs was shown by Key and Retzius[1] using a subarachnoid injection of gelatine containing Berlin blue, and later by Weed[4] with potassium

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† Eli Lilly Traveling Fellow, Medical Research Council of Great Britain.

ferrocyanide. Other substances such as India ink have been used for the same purpose. The perineural channels of the optic and olfactory nerves were demonstrated radiologically by Stuck and Reeves[3] using Thorotrast* and by Strain, French and Jones[2] using Pantopaque.†

In our preliminary studies we repeated the work of Strain, French and Jones,[2] and confirmed their observations. Five cc. of Pantopaque were injected into the cisterna magna of a dog under intravenous Nembutal anesthesia; the animal was then kept prone for 5 to 6 hours and x-rays were taken of the head at 30-minute intervals. The contrast medium passes in about 90 minutes into the fila olfactoria. These are arachnoidal tubes sur-

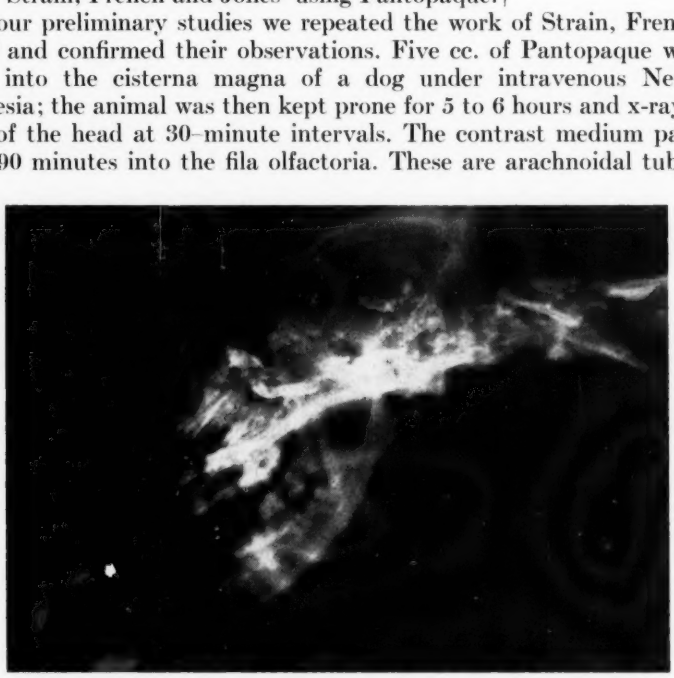

FIG. 1. Lateral view (dog) $6\frac{3}{4}$ hours after cisternal injection of 5 cc. of Pantopaque, with the animal prone. Contrast medium is seen in the optic nerve sheaths and in the fila olfactoria. Lymphatic glands behind the mandible are visible.

rounding the olfactory nerves which connect the olfactory bulbs through the cribriform plate with the submucous tissues of the nose. From the fila olfactoria the Pantopaque passes into the submucous spaces and into lymphatic channels through which it travels to lymph glands behind the mandible and in the neck (Fig. 1). The contrast substance also enters the sheaths of the optic nerves and passes into the epichoroidal and episcleral spaces from which it reaches the filtration angle of the eye and traverses the facial lymphatics to the anterior lymph glands of the neck[4] (Fig. 2). After 5 days most of the Pantopaque has left the nerve sheaths, and the remainder is scattered into droplets over the base of the cerebral hemispheres. The amount of Pantopaque seen there slowly decreases, and after 19 days the

* Colloidal Thorium Dioxide.

† Ethyl Iodophenylundecylate.

glands are almost free, only scattered intracranial droplets remaining which are still visible after 3 1/2 months. As in the experiments of previous investigators, the animal was in a prone position.

In our next studies a cisternal injection of 3 cc. of Pantopaque was given under intravenous Nembutal anesthesia with the animal supine and the head and neck extended. As expected, the Pantopaque passes over the surface of the cerebral hemispheres and along the borders of the superior sagittal sinus. However, it still enters the nasal tissues and optic nerve sheaths, although it now takes 5 hours to reach them. It is still visible in these situations 19 hours later (Fig. 3), after which it gradually disappears except for a few droplets over the cerebral hemispheres.

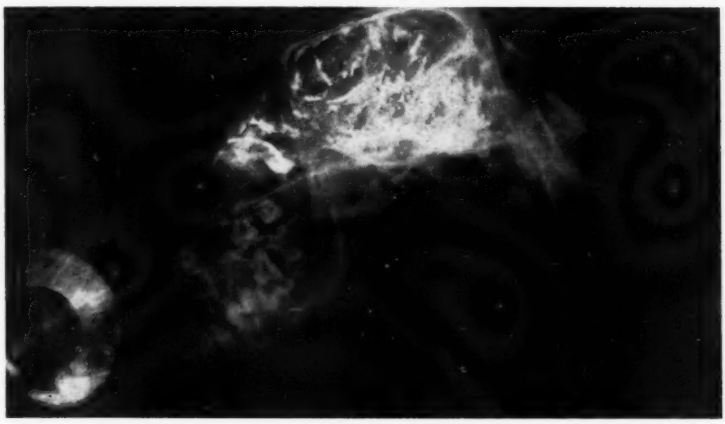

FIG. 2. Lateral view (dog) showing Pantopaque surrounding the globe of the eye 24 hours after cisternal injection. Anterior lymphatic glands are visible in the neck as well as those behind the mandible.

Comparison with Other Animals. In the cat, maintained in a prone position under intraperitoneal Nembutal anesthesia, it was found that the contrast does not enter the nasal tissues or optic nerve sheaths, although it flows into a suitable position for this to occur; the same thing was found in the kitten. However, in the Rhesus monkey it appears in the optic nerve sheaths in 15 minutes, although at no time does it enter the nasal tissues (Fig. 4).

Since Pantopaque is more viscous than CSF, these experiments were repeated in a cat and a monkey with Thorotrast, which has a relatively low viscosity. The results were the same except that a faint trace of contrast medium was seen in the optic nerve sheaths of the cat. These findings were in agreement with those of Stuck and Reeves[3] except that we did not observe any development of hydrocephalus in these animals after 4 months.

Significant differences therefore exist in the CSF circulations of these three animals. In each the arachnoidal villi are of importance, and in the cat and monkey they are certainly the chief route of absorption. In the dog,

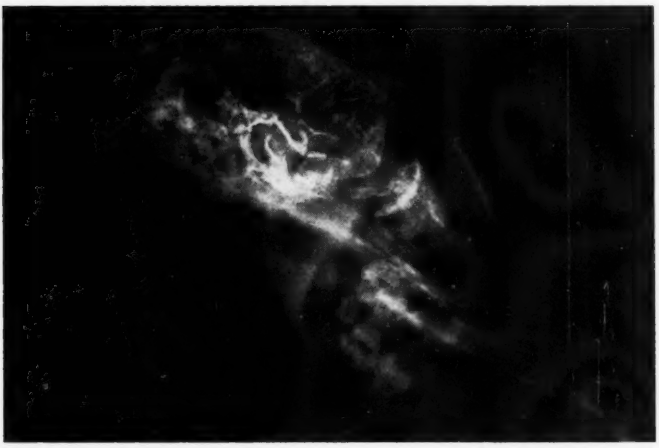

FIG. 3. Lateral view (dog) 24 hours after cisternal injection of 3 cc. of Pantopaque with the animal supine. Contrast substance is seen over the surface of the cerebral hemispheres, in the fila olfactoria, and in the sheaths of the optic nerves.

however, there are also important fluid exits through the nasal tissues and optic nerve sheaths, while in the cat the optic nerve sheaths are of but slight significance and the olfactory pathways of none at all. In the Rhesus monkey the optic nerve sheaths, but not the nasal channels, achieve an important role.

The Relative Importance of the CSF Pathways of the Dog. The effects of interruption of the main absorptive channels of the dog were investigated, in order to establish their relative importance.

Pantopaque will produce a fairly intense inflammatory reaction in the cerebral subarachnoid spaces of the dog (Fig. 5). This is confirmed during life by the failure of a second cisternal injection to pass into the olfactory and optic nerve sheaths. Instead, the Pantopaque is diverted by the basal obstructive process into the ventricles and over the cerebral hemispheres where the remaining absorptive pathways are occluded. That these barriers are also effective against the passage of

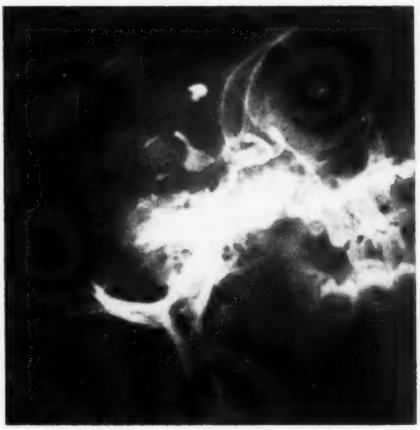

FIG. 4. Lateral view (Rhesus monkey) showing Pantopaque outlining the optic nerves 15 minutes after cisternal injection of 2.5 cc. Some of the contrast medium has also entered the 3rd and lateral ventricles. There is none in the nasal region.

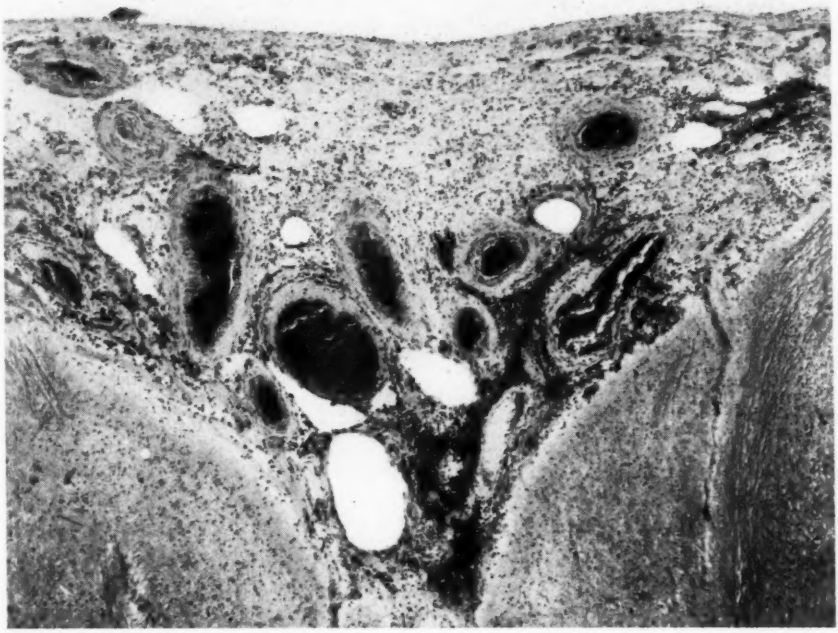

FIG. 5. Leptomeninges (dog) following cisternal Pantopaque injection. There is a zone of dense fibrous tissue beneath intact arachnoid cells. Between this zone and the pia the fibrotic reaction is largely, but not entirely confined to the periphery of the blood vessels. The lumens of the affected vessels are patent and enlarged. The muscular layers are intact and surrounded by wide zones of dense hyaline collagenous tissue. This perivascular fibrosis is the most distinctive feature of the meningeal reaction. The inflammatory cellular reaction is confined to scattered groups of lymphocytes and mononuclear cells, polymorphonuclear leucocytes being absent. (Hematoxylin and eosin)

CSF is shown by subsequent progressive ventricular enlargement and elevated intracranial pressure. The effect is the same whether 3 1/2 months or 7 days separate the 2 injections (Fig. 6). This experiment was carried out in 5 dogs with the same findings each time (2 dogs received 3 injections, but the ventricles were already enlarged when the third was given).

A single injection of 3 cc. of Pantopaque will not alone induce hydrocephalus (3 control animals) whether the animal has been kept prone or supine after it has been given. This indicates that if most of either the perineural or villous pathways are occluded the others are still sufficient to absorb enough CSF to prevent symptoms and signs of obstruction. The volume of Pantopaque used is not the operative factor, since there was no enlargement of the ventricles of a dog 5 weeks after a cisternal injection of 6 cc. of the substance.

Obstruction in the interpeduncular and neighboring cisterns was produced in dogs by the following method. A polyethylene tube was introduced

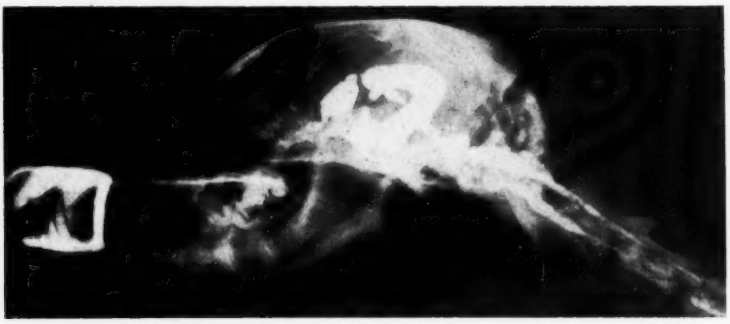

FIG. 6A. Lateral view (dog), showing enlargement of the ventricles 1 week after 2 cisternal injections of 3 cc. of Pantopaque. No contrast has entered the fila olfactoria or optic nerve sheaths (see Fig. 6B).

into the interpeduncular region through a subtemporal craniotomy under intravenous Nembutal anesthesia. The animals were then kept in a prone position while injections of a suspension of 250 mg. of kaolin* in 2 cc. of normal saline were given in 0.5 cc. doses over a period of 3 to 4 hours. It does not matter if some of the kaolin enters the subdural space. A subarachnoid fibrosis occurs which prevents the forward passage of CSF beyond the obstruction (Fig. 7). With kaolin a diffuse inflammatory response is produced, in contrast to the response to Pantopaque which is concentrated in zones beneath the arachnoidal surface and especially about the blood vessels.

FIG. 6B. Submento-vertical view (see Fig. 6A).

Obstruction of the interpeduncular cistern of 12 dogs by this method caused only a minimal degree of ventricular enlargement or none at all. This indicated, as in our previous experiments, that a satisfactory alternative route of absorption still remained. The basal obstruction then prevented a subsequent cisternal injection of Pantopaque from entering the sheaths of the olfactory and optic nerves of 7 dogs. Following the Pantopaque injection, the remaining pathways were occluded by inflammatory reaction in the meninges and hydrocephalus resulted (Fig. 8).

Obstruction of the ambient cistern has been produced by means of a suspension of kaolin introduced through a tube in 2 dogs, and the effects were similar to those of interpeduncular obstruction.

* Hydrated Aluminum Silicate.

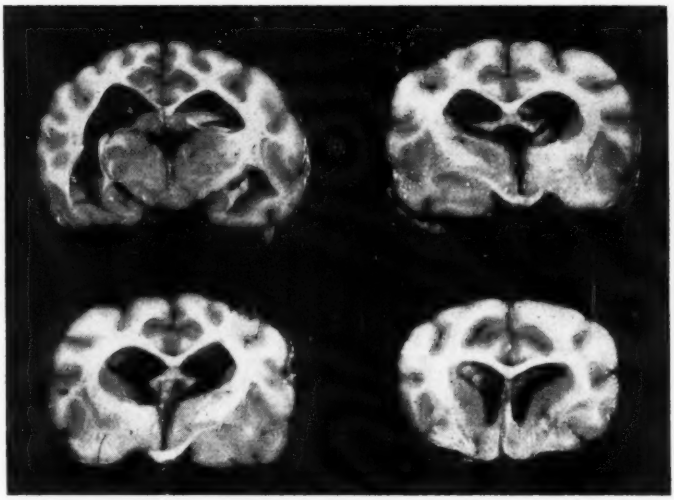

FIG. 6C. Brain slices showing the degree of hydrocephalus in the same animal as in Figs. 6A and B, 4 months after injection.

The olfactory CSF pathways were divided surgically in 2 dogs. No ventricular enlargement because of deficient absorption occurred. One of these received an injection of 3 cc. of Pantopaque into the cisterna magna; this was followed by enlargement of the ventricles—a hydrocephalus which was demonstrated to be communicating in type by the intraventricular injection of phenolsulphonphthalein (PSP) which appeared at the cisterna magna in 30 seconds.

Obliteration of 50 per cent of the subarachnoid pathways over the surface of the cerebral hemispheres can be brought about by an arachnoiditis induced by covering the brain with cellophane or iodinated gauze; this causes a very dense fibrosis of the meninges. Despite this reduction of absorptive channels, there are no signs of hydrocephalus until the optic and olfactory routes are also obstructed by a Pantopaque arachnoiditis. When this occurs hydrocephalus rapidly develops.

CONCLUSIONS

In the dog there are three main paths of CSF absorption: the arachnoid villi leading to the blood stream, and the perineural channels of the optic and olfactory nerves leading to the lymphatic system. Weed[4] using ferrocyanide found granules of Prussian blue from the vagus nerve sheath in the middle of the neck; from the spinal accessory sheath they traveled 1 to 2 cm. and from the hypoglossal nerve sheath they reached the tongue. However, these experiments were done under raised pressure, and we consider that the contribution of nerve sheaths other than the optic and olfactory is not significant. Each of the three main routes described appears to be of almost equal importance in the dog, a state of affairs that does not exist

FIG. 7. Leptomeninges (dog) following injection of kaolin suspension, which is seen as fine (brown) granules; they are filled diffusely with the material and the reaction to it. Large, sometimes discrete masses of mononuclear cells are crammed with phagocytosed kaolin particles, and among them are lymphocytes; giant cells are rare. This inflammatory cellular infiltration is associated with the growth of collagenous connective tissue. Groups of polymorphonuclear leucocytes show the continuing nature of the reaction. Extensive proliferation of fibrous tissue within and beyond the zone of reactive cells fills the subarachnoid space, forming a sheet of connective tissue with focal granuloma-like masses scattered through it. (Hematoxylin and eosin)

in the cat or Rhesus monkey. This amplifies the differences among these animals which we earlier demonstrated radiologically. There is reason to believe that similar differences exist between the CSF circulations of dog and man. Great care must therefore be exercised in drawing conclusions based on evidence from the lower animals about this circulation in man, and it follows that the dog is not suitable for comparative experimental work on the absorptive aspect of the CSF circulation.

In view of the inflammatory reaction that we have seen with Pantopaque,

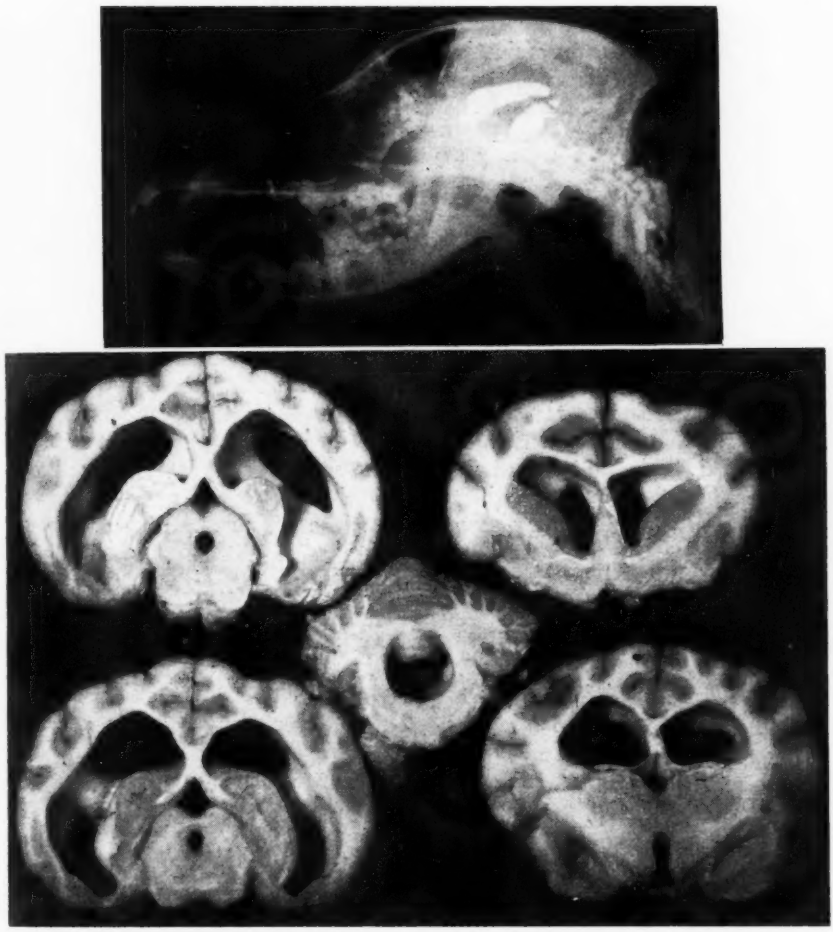

FIG. 8. (*Upper*) Lateral view (dog) 28 hours after a cisternal injection of 3 cc. of Pantopaque given 3 weeks after obstruction of the interpeduncular cistern with kaolin. No contrast medium has passed beyond the obstruction in a forward direction. The ventricles are outlined and show slight enlargement. (*Lower*) Brain slices of the same animal showing the degree of hydrocephalus 2 months later.

we must emphasize that we do not consider it unsuitable for clinical use in myelography. In our experiments we employed a greater amount relative to the total CSF volume than is used clinically. However, our findings show the need for removing as much as possible and for keeping it away from the cerebral subarachnoid spaces, since it causes subarachnoid fibrosis when used in dogs.

EXPERIMENTAL COMMUNICATING HYDROCEPHALUS

In the course of our experiments various types of obstruction of the CSF

pathways have produced enlargement of the ventricles. This hydrocephalus is almost always of communicating type.

Five dogs were each given 2 cisternal injections (2 dogs had 3 injections) of Pantopaque, one prone and the other supine. The basal pathways and the channels over the cerebral hemispheres were thus obliterated. We found 1 or 2 weeks to be a suitable interval between injections. In all these animals communicating hydrocephalus developed. After 3 injections in 1 animal a block developed in the cisterna magna, which later re-opened. Three control animals received only 1 injection and did not become hydrocephalic. There were no deaths in this series.

Seven animals received interpeduncular kaolin suspension followed by cisternal Pantopaque. Two weeks elapsed between the 2 procedures. In 6 of these communicating hydrocephalus developed which was of considerable degree in 5 (Fig. 9). There was 1 death 4 days after Pantopaque injection, the cause of which was not clear. Proof of communication was afforded by combined cisternal and ventricular taps, with or without the injection of PSP or other dye. This method has not been so successful in Rhesus monkeys; hydrocephalus developed in only 1 out of 3.

In 2 dogs that received cisternal injections of 2 cc. of Lipiodol* com-

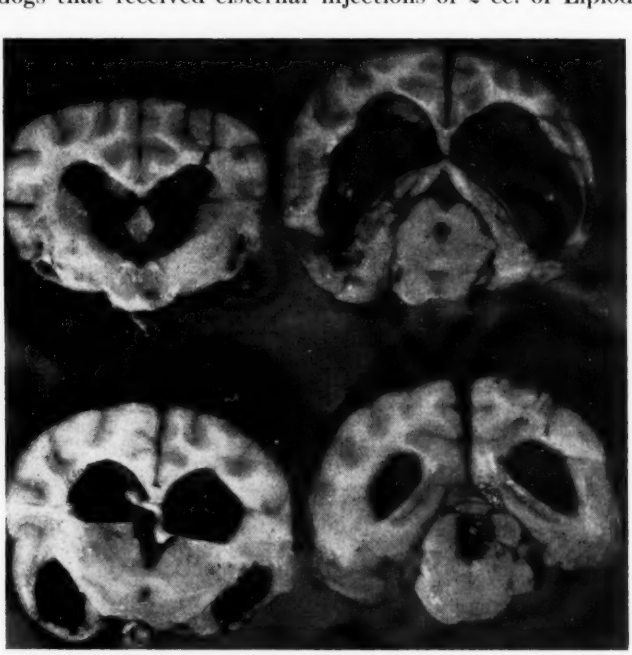

FIG. 9. Brain slices (dog) showing considerable ventricular enlargement 1 month after occlusion of the remaining subarachnoid CSF pathways with Pantopaque following interpeduncular obstruction with kaolin.

* Lipiodol Lafay.

municating hydrocephalus developed but the rate of development was too slow for the method to be of experimental use.

In the animals in which the olfactory pathways were divided or the cerebral hemispheres covered to produce fibrosis, communicating hydrocephalus developed, but owing to the extent of the operative damage the preparation would be unsuitable for some experiments.

Two satisfactory methods of producing communicating hydrocephalus have therefore been found. However, after 2 injections of 3 cc. of Pantopaque a small amount of the contrast medium remains in the CSF and cannot be absorbed, at least for several months, whereas following an interpeduncular injection of kaolin hydrocephalus may be produced with as little as 1 cc. of Pantopaque. This has almost all disappeared by the time hydrocephalus has developed to a satisfactory degree, which takes about 3 weeks.

Interpeduncular injection of kaolin suspension followed by cisternal injection of Pantopaque is therefore the most satisfactory method for the experimental production of communicating hydrocephalus. The technique has been used chiefly on dogs, but since the absorptive channels are obstructed, the differences in CSF absorption between dogs and other animals are not a consideration when using it for comparative studies.

SUMMARY

1. Significant differences in the mechanisms of absorption of CSF are shown in the dog, cat and Rhesus monkey by following the course of a cisternal injection of Pantopaque.

2. Pantopaque will produce arachnoidal fibrosis when injected into the subarachnoid space of the dog.

3. Obstruction of arachnoid villi, and the perineural subarachnoid pathways in the dog, show both to be of importance in the absorption of CSF in this animal. This is not true in the cat or Rhesus monkey.

4. Care should be exercised in comparing the CSF absorption mechanisms of lower animals with those of man.

5. Methods of producing experimental communicating hydrocephalus in the dog are described.

We are indebted to Dr. Orville T. Bailey for reports on the microscopical sections.

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NEUROLOGICAL SURGERY

ITS PAST, PRESENT AND FUTURE*

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I WISH to express my deep appreciation of the honor of serving as your president this year. May I thank you also, at this time, for the great privilege of being one of your representatives on the American Board of Neurological Surgery during the past six years. This address will be, in part, an accounting of my stewardship to you. However, I would like it to be more than that. With this meeting the Harvey Cushing Society has "come of age;" it is twenty-one years old. It has acquired responsibilities as well as size and maturity. As the largest organization of neurological surgeons in the world, the responsibility for the future of the art-science to which we are dedicated is ours. The principal means of projecting the present into the future is by training those who will follow. We all owe such a debt to the past, which we must pay to the future. As I wrote this I suddenly realized that I was, generation-wise, between the present and the past; and, in the words of Plutarch, "It was hard for him who had lived in one generation of men to plead before another."

But I will plead my case, for it is a good one. The case is neurological surgery, its past, present and future, and more particularly the American Board of Neurological Surgery.

THE PAST

For a glimpse of the field of neurological surgery in the 1920s we should start with a small gathering on March 19th and 20th, 1920, at the Peter Bent Brigham Hospital in Boston, when the Society of Neurological Surgeons was organized. Present at this meeting were Cushing, Frazier, Lewis, Sachs, Adson, Towne, Harvey, Horrax, Dowman, Bagley and Mixter. The purposes of the Society were: "1. The development of the field of neurological surgery. 2. The education of the medical profession and particularly the surgeons in the idea that neurological surgery requires a special training in addition to that of the general surgeon." There can be little doubt that these objectives have been in large part attained. Fig. 1 shows some of these pioneers at their meeting in 1922.

During the '20s young neurosurgeons were being trained in about fifteen centers throughout the country. By the early '30s "the idea that neurological

* Presidential Address, delivered at the meeting of the Harvey Cushing Society, Hollywood, Florida April 23, 1953.

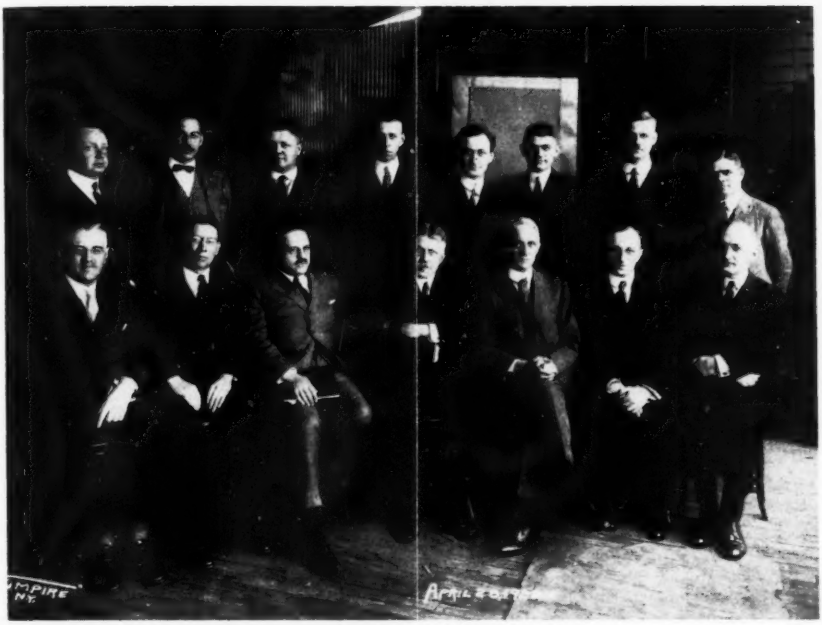

FIG. 1. Photograph of the Society of Neurological Surgeons taken at meeting in 1922.

surgery requires a special training" was being preached by being practiced. "Those fellows in the laboratories" of anatomy, physiology, pathology, the radiologist, ophthalmologist and neurologist were seen with increasing frequency in the company of the neurosurgeon. That a new team was being formed was evident by 1932, when some of this "second generation" appeared in the operating room of the Peter Bent Brigham Hospital on the morning of May 6, 1932, to watch the "Chief" do a third ventricle tumor. Later, at the business meeting in the auditorium of the Children's Hospital (and not without labor pains) the Harvey Cushing Society was born. Its object was: "The promotion and advancement of neurological surgery." This is followed in the Constitution by the following pertinent statement: "The past accomplishments in neurological surgery have been effected through the close cooperation of all those interested in the science of neurology and its collateral branches. The specialized field of neurological surgery can be further advanced by close cooperation of these various groups."

The 1930s saw tremendous development in the field of neurological surgery and in the number of neurological surgeons. In April 1939 the Harvey Cushing Society gathered in New Haven for the 70th birthday party for the "Chief." A glance at a photograph (Fig. 2) taken on that memorable occasion will confirm that this Society as well as the specialty had grown up.

FIG. 2. Photograph taken at the meeting of the Harvey Cushing Society in 1939.

Already there were gentle stirrings toward establishment of a certifying board in neurological surgery. The American Board of Ophthalmology had been activated in 1917, otolaryngology in 1924. By 1938 a total of fourteen specialty boards had been formed. Hospitals were beginning to require board certification as a qualification for staff appointments. The younger neurosurgeons wanted a certifying board in neurological surgery, although some had already been certified in surgery or neurology. At the meeting of the Society of Neurological Surgeons in June 1938 the organization of such a board was approved. This was followed by similar action by the Harvey Cushing Society. After preliminary meetings in November 1939 and April 1940, the American Board of Neurological Surgery (Table 1) was officially activated October 17, 1940, in Chicago. Its stated purposes are: "To encourage the study, improve the practice, elevate the standards and advance the science of neurological surgery and thereby to serve the cause of public health." This Society can be of inestimable help in attaining those objectives; it is my fervent hope that we will.

Board members are appointed for terms of six years by the following societies: The Society of Neurological Surgeons (4), Harvey Cushing Society (3), American Academy of Neurological Surgery (1), American College of Surgeons (1), American Neurological Association (1), American Medical Association, Section on Surgery (1), American Medical Association, Section

TABLE 1

American Board of Neurological Surgery
(1940–1953)

First Board Members	
Naffziger (Chairman)	
Adson (Vice-Chairman)	
Spurling (Secretary-Treasurer)	
Bucy	*Fay
*Craig	Peet
*Davidoff	Putnam
Davis	Sachs
	Stookey
Interval Members	
Bailey	*German
Coleman	Horrax
Evans	Semmes
	Van Wagenen
Present Board Members	
Grant (Chairman)	
Spurling (Vice-Chairman)	
*Furlow (Secretary-Treasurer)	
Brown	Oldberg
Campbell	Ray
Cone	*Walker
*Ingraham	White
	Wilkins

* Appointees from the Harvey Cushing Society.

on Neurology and Psychiatry (1). There have been a total of thirty men on the Board to date. Examinations have been held at least once a year, with a total of nineteen examinations, in the following cities: Chicago, Philadelphia, St. Louis, New York, San Francisco and New Haven.

Up to the present there have been 403 successful candidates. In addition, 56 "individuals of professorial rank" were certified on invitation by the Board, without examination. Thus, in the twelve years of its existence the Board has certified 459 neurosurgeons (Fig. 3). In 1951 a special foreign

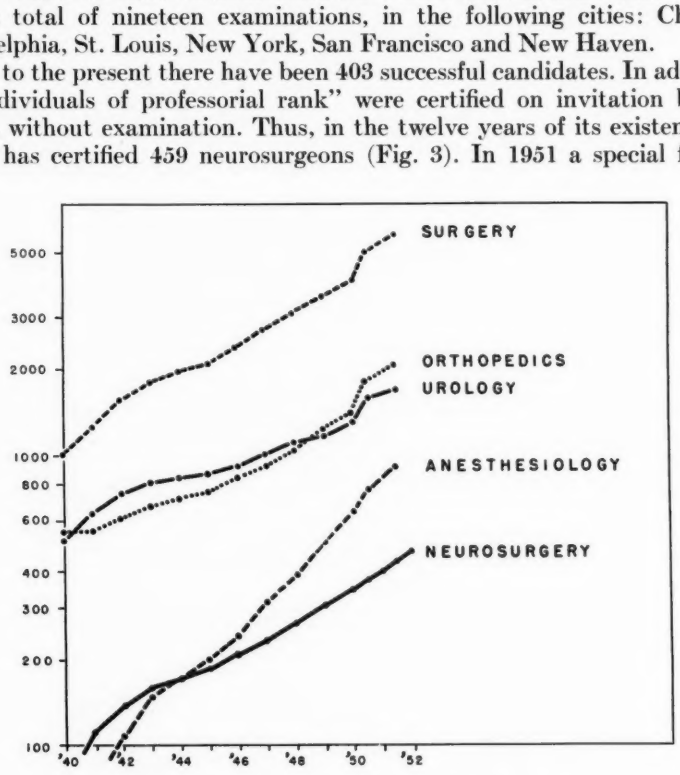

FIG. 3. Log curve of diplomates certified, 1940–1952.

certificate was offered by the Board to foreign candidates who have received their training in neurological surgery in the United States or Canada and who are returning to their own country at the end of their training period, upon passing successfully the regular examinations of the Board, *without* completion of the usual requirement of two years in the practice of neurological surgery. This was done with the aim of promoting international understanding and good will and of sharing our training advantages with others. Three diplomates have been certified on this basis. All told, there are nine diplomates practicing in foreign countries. Of the remaining 450, nine are deceased and nine are inactive in neurological surgery, leaving a present total of 432 active diplomates in the United States and Canada.

During the first few years of the Board's existence it became apparent that information concerning the various training programs was essential for

he proper evaluation of the candidates' credentials. In 1945 the Board ndertook to act in an advisory capacity to the Council on Medical Edu- ation and Hospitals of the American Medical Association in the approval f residency training programs in neurological surgery. This activity has ecome almost as important as the certification of diplomates. In 1948 here were 57 approved institutions offering 138 residencies and fellowships. y 1950 the figures had risen to 79 and 181 respectively. The 1952 listing ncludes 94 approved institutions with 241 traineeships. However, in the cademic year 1951–52 there were 17 per cent vacancies in neurosurgical esidencies, with 38 per cent vacancies at the first-year training level.

About two years ago the Board took cognizance of the ancient proverb, A time for fishing; a time to mend the nets." It began to inspect the nets y instituting a statistical survey of its activities during the years of its xistence. A considerable bulk of pertinent data has been assembled, but I ill briefly mention only the following items: Where do neurosurgeons come rom? Where do they train? Where do they practice? How do they get long in their Board examinations?

Neurosurgeons come from everywhere, as illustrated by the fact that hose accepted for examination included graduates of 67 medical schools in he United States and Canada; in addition, there were 15 graduates of oreign medical schools. However, if we exclude the foreign graduates from he calculation, about half of the neurosurgeons came from the eleven medical schools listed in Table 2.

TABLE 2

Medical schools from which neurosurgeons were graduated

Harvard	35	Yale	14
Johns Hopkins	31	McGill	11
Pennsylvania	21	Vanderbilt	11
Michigan	20	Cornell	10
Chicago and Rush	16	Illinois	10
		Northwestern	10

Since most candidates had their neurosurgical training in more than one institution, we might think they train everywhere. There are, indeed, 124 training institutions represented. But over half of the total training was obtained in the sixteen training programs listed in Table 3.

By this time you are probably willing to accept that they practice everywhere. That is a reasonable approximation, but not quite accurate.

At present there are 409 active, certified neurosurgeons in the United States, excluding those in the regular Army and Navy. This gives an overall concentration of .27/100,000 population (Fig. 4). An acceptable estimate for 1954 is 520, or .33/100,000. Nine states (Conn., Mass., Vt., Tenn., Ariz., Minn., Colo., Calif., Nev., and D.C.) already have .4/100,000 or over, that is, 1 per $\frac{1}{4}$ million population (Table 4).

TABLE 3

*Neurosurgical training programs**

Mayo Clinic	38
Montreal Neurological Institute	25
New York Neurological Institute	24
Illinois Neuropsychiatric Institute	15
Yale University—Hartford Hospital	15
University of Michigan	13
Lahey Clinic	12
Long Island College	11
Boston City Hospital	10
University of California	10
University of Chicago	10
Johns Hopkins University	10
University of Pennsylvania	10
Barnes Hospital	9
Brigham—Children's Hospital	9
Los Angeles County Hospital	9

* The figures represent trainee equivalents of candidates who have taken the Board examinations through 1952, corrected for split-training in various institutions.

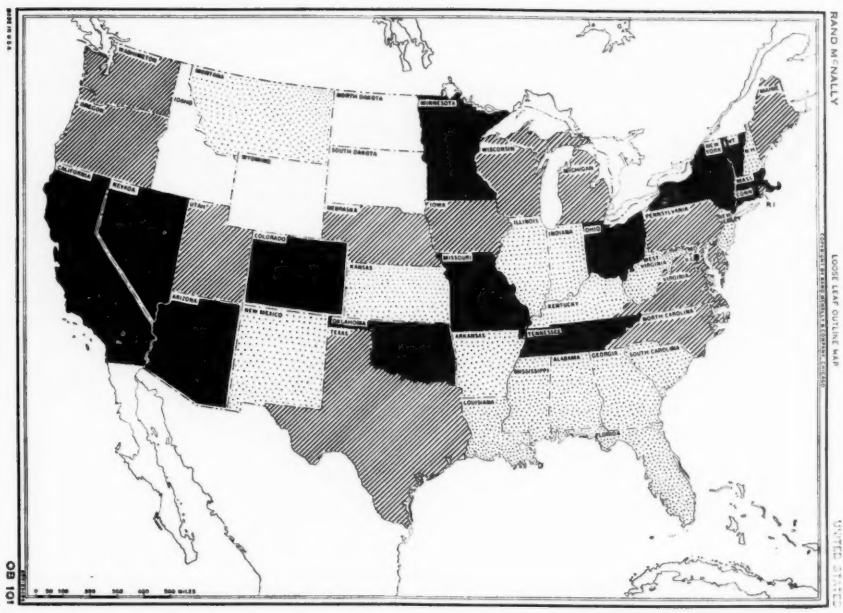

FIG. 4. Geographical location of active certified neurosurgeons in the United States, excluding those in the regular Army and Navy. *White:* 0. *Stipple:* less than .22 per 100,000 population. *Hatched:* .22 to .31 per 100,000. *Black:* greater than .31 per 100,000. These figures are as of April, 1953.

TABLE 4

Active certified neurosurgeons per 100,000 population, April, 1953

New England			Indiana	.13	.18*
Connecticut	.5		Michigan	.23	
Maine	.22	.33*	Ohio	.37	.38*
Massachusetts	.4		Wisconsin	.24	
New Hampshire	.2	.4*	*West North Central*		
Rhode Island	.25		Iowa	.23	.27*
Vermont	.5		Kansas	.05	.16*
Central Atlantic			Minnesota	.4	.43*
Delaware	.3		Missouri	.37	
District of Columbia	1.0		Nebraska	.3	
Maryland	.26		North Dakota	0	
New Jersey	.12	.17*	South Dakota	0	
New York	.35	.36*	*South West*		
Pennsylvania	.24	.25*	Arizona	.4	
West Virginia	.15		New Mexico	.14	
South East			Oklahoma	.32	
Alabama	.16		Texas	.26	.28*
Arkansas	.05	.16*	*Rocky Mountain*		
Florida	.21	.29*	Colorado	.46	
Georgia	.2		Idaho	0	
Kentucky	.2		Montana	.17	.34*
Louisiana	.11	.15*	Utah	.29	
Mississippi	.05		Wyoming	0	
North Carolina	.22		*Far West*		
South Carolina	.05		California	.42	.44*
Tennessee	.42		Nevada	.5	
Virginia	.27	.3*	Oregon	.27	
East North Central			Washington	.29	
Illinois	.21	.22*			

* Corrected for diplomates certified June, 1953.

Now, about examination results. These have been correlated with the various residency training programs. An interesting accumulation of information has resulted, but its interpretation is outside the scope of this report. There is now a need for some means of correlating the results of examinations with the abilities demonstrated by the diplomates in the years following

TABLE 5

Board examination grades

Highest	*Subject*	*Failure*
27.0%	Neurosurgery	9.1%
20.7	Neurology	11.9
25.0	Anatomy and Physiology	8.4
17.6	Pathology and Bacteriology	9.5
23.5	General Surgery	5.3
20.2	Ophthalmology	6.4
21.4	X-Ray Diagnosis	5.5
22.2%	Average	8.0%

examination. I am sure the Board would welcome your suggestions on this matter. It is possible now, however, to present an overall picture of examination results, based on the highest and lowest marks obtained in the various subject-examinations during the Board's existence (Table 5). Over these years 26.3 per cent of the candidates have failed in one or more subjects on their first attempt. Most of these eventually passed.

THE PRESENT

PROFILE OF A NEUROSURGEON

Age. In 1950 the median age of certified neurosurgeons was 45. The average for diplomates of all boards was 48, our specialty being exceeded in juvenility only by anesthesiology (Table 6).

TABLE 6

Diplomates' median ages[2]

Anesthesiology	43
Neurosurgery	45
Surgery	46
Orthopedics	47
Urology	51
Otolaryngology	54
Average	48

Activities. About 170 neurosurgical diplomates are engaged in some aspects of residency training programs. Seventy-eight are chiefs of university divisions of Neurological Surgery. Twenty are in "clinics," 11 in the Veterans Administration and 10 in the Army or Navy.

It appears that what we have all suspected is true: The neurosurgeon works harder than anyone else! In 1950, 102 neurological surgeons reported working an average of 65.2 hours per week. This was the highest of all groups reporting, the runner-up being pediatricians with 62.2 hours. The mean for all physicians was 58.3 hours; the lowest was 44.1 for dermatologists and syphilologists (Table 7).

TABLE 7

Average working hours per week[2]

Neurosurgery	65.2
Pediatrics	62.2
Obstetrics and Gynecology	61.1
General Practice	60.6
General Surgery	59.5
Medicine	59.0
Urology	54.6
Neuro-Psychiatry	50.5
Dermatology and Syphilology	44.1
Average	58.3

Income. This, we know can't be true. In 1949 neurological surgeons had the highest net income of all groups. Their *mean* net income was about $24,000, *before* taxes. This was about $9,000 more than the urologists, $15,000 more than the general practitioners, and $13,000 above the average of all groups (Table 8).

TABLE 8

Net income, 1949[2]
Mean to nearest $1,000

Neurosurgery	$24,000
Orthopedics	17,000
Radiology	17,000
General Surgery	16,000
Urology	15,000
Anesthesiology	12,000
General Practice	9,000
Average	11,000

From the foregoing it would appear that the present neurological surgeon is a young man who is overworked and overpaid. He has a little-better-than-even chance to retire before he dies. A short time ago Professor Olivecrona visited this country. Before leaving he attended a meeting of the New England Neurosurgical Society, where he was asked if he would be willing to give a few words of advice to the young neurosurgeons present. He said he would; that he had visited many sections of our country and many of our institutions. He concluded, "My advice is this: Don't work so hard."

THE FUTURE

The previously cited factors, plus the present momentum of young men toward neurological surgery indicate probable growth by 1960 to about 800 active neurosurgical diplomates in the United States, representing slightly less than 1 per 200,000 population (.47/100,000).

Training Problems. Present approved training programs are capable of turning out almost 500 potential diplomates for certification by 1960. Since only 8 per cent of neurosurgical diplomates were over 54 years old in 1950, a large proportion of the present team will still be on the field by 1960. After allowances for wear and tear of the years and diplomates residing outside the United States, this would result in almost 800 active certified neurological surgeons in the United States by 1960, or almost double the present figure of 409. It appears obvious, therefore, that there is no urgency to expand the number of residency training programs. In fact, the previously cited 38 per cent vacancies in first-year neurosurgical residencies in the year 1951–52 confirm that there is no demand for such expansion. However, when the numbers of approved training programs and residencies are log-plotted for the interval 1948–52, the curves parallel the slope of the diplomates' curve very closely. If the increase of approved training programs

were to continue at the present rate, there would be 250 programs offering 650 residencies by 1960. This is obviously ridiculous.

Furthermore, there is already evident a considerable dilution of clinical material for resident training in existing neurosurgical centers. It is doubtful that there are over 50 training programs in the United States where the senior resident will actively participate in the care of 50 brain tumors per year. However, the pressure for approved programs will continue: "Everyone wants a resident." I believe that the Board has a manifest responsibility to resist this pressure, unless we wish to train two types of residents: a few "tumor-surgeons" and many "disk-surgeons." One hundred approved training programs would afford ample opportunity for trainee choice and healthy competition. Reappraisal of each program every three years, as presently practiced, will permit appropriate changes within the approved list; always with the objective of improving the quality of training.

The large ward services, which were the chief source of clinical material for residency training, have been greatly reduced in the country at large during the past ten years and the present unstable financial condition of many of our hospitals presages further reduction. Conversely, hospital admissions have increased, especially in the semi-private category. The reason for this is not hard to find. In Connecticut, approximately half the population is covered by *Blue-Cross* and a third by the *Connecticut Medical Service* plan. Appropriate means must be found to take maximum advantage of this clinical material for our training programs. Residents in university hospitals usually have faculty appointments as instructors. The substitution of the traditional long white faculty coat in place of the house officer "whites" would be in keeping with their rank and experience. To a large segment of the public, all men in white are internes, and no one wants to be operated upon by an interne. Few object to being cared for by an instructor, with the professor in an advisory role. Similar modifications of the status of senior residents in "clinics" and non-university hospitals should not encounter insurmountable obstacles.

Further broadening of the training base may be obtained by the use of joint-training programs. For example, our own program in Connecticut is based upon a university and the two largest hospitals in the State with the new Veterans Administration Hospital soon to be added. It encompasses 10 of the 13 certified neurosurgeons in the state.

Board Requirements. I agree completely with the ideas expressed by Doctor Bucy[1] in his presidential address to this Society last year: "It would be unwise to diminish the present flexibility of the training requirements." Rigidity does not promote quality, but discourages initiative and individuality. However, the present requirements should be clearly recognized as the absolute minimum, not the ideal. As long as the need for a considerable number of physicians in the military services continues, I believe it would be undesirable to increase the training time required by the Board. When and if this obligation is resolved, the addition of an "elective" year might be considered. This could be spent in neurological surgery, general

surgery, neurology or the basic sciences and would solve the present "excess" year of training required of candidates from states in which rotating interneships are obligatory. Already the following Boards have adopted four-year training requirements: Orthopedics, Pathology, Plastic Surgery and Surgery. Only Anesthesiology and Pediatrics require less than three years beyond the interneship.

The question of actual surgical experience of the candidate, in the role of operator, has been a matter of special interest to the Board recently. The various shades of opinion have been argued long into the night. However, the philosophy inherent in the problem may be expressed most succinctly in the words of Sam Harvey: "Where residents do a considerable amount of operating, the institution's mortality will be a little higher, but the overall life-time mortality of the trainee will be lower."

Board Examinations. I have worked with all the examining teams of the Board during the past six years and am completely convinced of the absolute fairness of the examiners without exception. I can imagine no problem in this respect in the future. All subjects lend themselves to the types of examinations that are given, with the possible exception of the most important subject: neurological surgery. Judgment, resourcefulness, "a nose for a tumor or abscess," the ability to keep out of trouble, are difficult to assess in an hour examination. And, how do you examine a man on trigeminal root section when he tells you that he has never done one? He may even describe the operation perfectly, but, is that enough? This naturally leads to the ultimate question: What does Board certification mean? What *should* it mean? A majority of the certified neurological surgeons of this country are in this room. The answer is yours to make!

But the answer must be approached very cautiously. Ours is but one of a number of Specialty Boards. The answer should be one acceptable to all and applied by all Boards, after previous negotiation through the Advisory Board for Medical Specialties. Perhaps the answer for our specialty would be to give a candidate his "first papers" after passing the present type of examination, adding a final comprehensive examination in neurological surgery, for full or special certification, after five or ten years of satisfactory practice. The problem is a thorny one. Hospitals would have to be educated to the necessity of allowing full practice privileges to the candidates during the interval between examinations. The plan would have to be so devised that it could not be interpreted as a measure "in restraint of trade." Nor should any move be made that would discourage the entry of talented young men into our field. It need not; the challenge would be greater, the goal though more distant, more worthy of attainment. And the Board, and all of us could say, when a man finished: "There goes a neurological surgeon."

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CASE REPORTS AND TECHNICAL NOTES

TRAUMATIC ARACHNOIDAL CYST

SIMULATING ACOUSTIC NEURINOMA

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A review of the literature on arachnoidal cysts in the posterior fossa reveals 34 cases reported to date.[1–4] In all of these cases the cysts occupied a posterior position in relation to the cerebellum, 5 of them being midline posterior. The cyst in the present case differs from the rest in being located anteriorly in the cerebellopontine angle.

Etiologically, these cysts are known to occur concomitantly with congenital malformations of the hindbrain,[2] are associated with local and distant inflammatory or infectious processes, and occur as a complication of blood in the subarachnoid spaces. Trauma, direct or indirect, as noted by Thompson,[4] may be an inciting factor. The case presented here certainly supports this view.

CASE REPORT

An 18-year-old colored male was admitted to the University Hospital Dec. 4, 1952. Three years previously he had fallen from a wagon and was rendered unconscious for 20 to 30 minutes. From this time on he carried his left arm in a semiflexed position. Shortly after the injury he began having severe, intermittent, frontotemporal headaches and difficulty in maintaining equilibrium. One year later he noted diminution in hearing in the right ear which was followed shortly by a greater loss of hearing in the left ear. Two years later weakness developed in the left arm and leg. Shortly before admission numbness and weakness developed in the

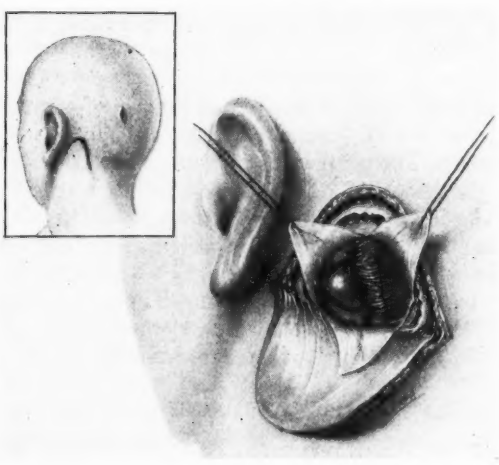

FIG. 1. A blue opaque cyst compressing the hemisphere medially and posteriorly

left side of his face and he could not walk unaided. He had diplopia and became drowsy to the stage of light stupor.

Examination. Positive neurological findings were bilateral papilledema of 2 D., bilateral abducens paralysis, hypalgesia in the distribution of the trigeminal nerve on the left with absence of the corneal reflex, left peripheral facial paralysis, and nerve type deafness in the left ear. Horizontal nystagmus was present on either lateral gaze. The left arm and leg were paretic. The Babinski sign was present on the left. The gait was wide based and markedly ataxic.

Roentgenogram of the chest was normal; that of the skull showed demineralization of the left petrous ridge.

Surgical exploration was decided upon on the basis of the clinical findings.

Operation. On Dec. 11, 1952, using hypotensive anesthesia, the left lateral ventricle was tapped and was found to be dilated by ventricular estimation. A left cerebellar angle exposure was made. The left cerebellar hemisphere was under increased tension. As the hemisphere was retracted medially a blue domed opaque cyst was disclosed anteriorly and laterally (Fig. 1). This was in a position similar to that seen over an acoustic tumor. An opening was made in the cyst wall, through which clear colorless spinal fluid escaped under pressure. Following this the cyst collapsed and the previously compressed hemisphere fell toward the midline (Fig. 2). The left cerebellopontine angle was then widely exposed without need of retraction. All of the nerves emerging from the pons and medulla could be seen through and around the now translucent cyst wall. The 5th nerve was stretched anteriorly and superiorly, and the 7th and 8th nerves were stretched anteriorly. The anatomy in this region was easily visible and no evidence of a tumor could be found. The cyst had markedly indented the anterolateral surface of the cerebellum.

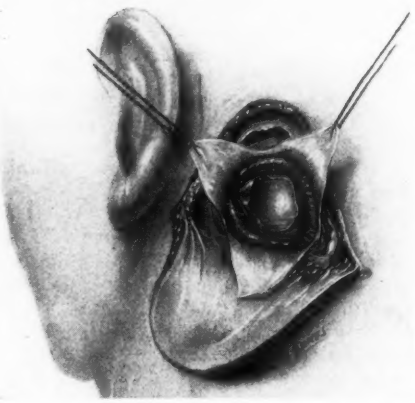

FIG. 2. After collapse of the cyst, the 5th, 7th and 8th cranial nerves could be seen through the anterior wall.

Postoperative Course. The diplopia and headache disappeared immediately. By the 3rd day the trigeminal and facial involvement had cleared and there was marked improvement in the motor power of the left arm and leg. One week after operation he was fully ambulatory with complete disappearance of the gait difficulty. The papilledema had receded and the auditory acuity was grossly normal. One month postoperatively there were no neurological abnormalities.

SUMMARY

The subject of arachnoidal cysts in the posterior fossa is reviewed. The present report adds a case in which the cyst was in a location not previously described. Trauma as a definite etiological factor in this case is presented.

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PITUITARY CYST

LINED WITH A SINGLE LAYER OF COLUMNAR EPITHELIUM

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Clinically cysts found within the hypophyseal fossa occur most commonly as the result of degeneration within a pituitary adenoma. Intrasellar cysts lined by a single layer of epithelium and large enough to give rise to signs and symptoms are very infrequent. Clinically they may be indistinguishable from chromophobe pituitary adenomas. The limited number of cases that have been reported is insufficient to clarify the symptomatology and they were not followed long enough to determine the prognosis with cysts of this type.

CASE REPORT

D. McL., a 41-year-old white male contractor, was admitted to The Chicago Memorial Hospital on March 21, 1948, and discharged on March 31, 1948. He was referred by Dr. Isabelle McGarry of Evanston, Illinois.

He was first seen in consultation on April 28, 1947. He had been complaining for 2 months that his eyes tired easily while driving. Because of this symptom he had consulted Dr. McGarry who found the visual acuity of the right eye to be 20/40, and of the left eye, 20/20. A complete upper temporal quadrantic defect was demonstrated in the right eye and a partial upper temporal quadrantic defect in the left eye. No other neurological symptoms nor any symptoms related to abnormal function of the hypophysis or hypothalamus were elicited at that time.

Examination, April 28, 1947. The patient was a fully oriented, alert, cooperative, obese white male. He weighed 225 lbs. and was 5 ft. 8 $\frac{3}{4}$ in. in height. B.P. was 170/100. His beard was sparse and the facial skin was finely wrinkled. The skin of the body was soft and smooth. Examination of the visual acuity and fields confirmed Dr. McGarry's findings. There was a definite pallor of both optic discs. The pupils were round, regular and equal and reacted well to light and in accommodation. The extra-ocular movments were full.

Roentgenograms of the skull revealed that the sella turcica was greatly expanded in all directions with marked thinning of the floor, which protruded downward into the region of the sphenoidal air cells. There was marked decalcification and thinning of the posterior clinoids. The pineal gland was calcified and showed a questionable displacement to the right.

Basal metabolism rate was −22.6 per cent. Glucose tolerance was within normal limits.

Clinical diagnosis: Pituitary adenoma of chromophobe type.

Course. In view of the patient's relatively good vision X-ray treatment was directed toward the hypophysis. He received two courses of 1500 R each, over a 10-day period at a 7-month interval. In spite of this therapy the defects in his visual fields increased although the visual acuity remained unchanged. In December 1947 a repeat BMR was −37 per cent. During the same month the patient began to notice a diminution of his libido.

Roentgenograms of the sella turcica in March 1948 showed no changes from the films taken a year earlier.

Because of the progression of the defect in the peripheral visual field and the decreasing

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libido the patient was admitted to the neurosurgical service of The Chicago Memorial Hospital.

Examination, March 21, 1948. The patient was obese, with scanty beard and soft and smooth skin. B.P. was 140/70. Visual fields demonstrated complete upper temporal field defects bilaterally. Visual acuity of the left eye was 20/30 and of the right eye 20/20. There was still definite pallor of both optic discs. There were no other neurological abnormalities. Blood count, urinalysis, and Wassermann and Kahn tests gave normal findings.

Operation. On March 22, 1948, a right frontal osteoplastic flap was reflected under local anesthesia. The optic chiasm was exposed by an extradural approach under the frontal lobe. The right optic nerve was pushed upward, compressed and thin. The chiasmatic cistern was opened exposing a very thin-walled cyst filling the sella turcica. The wall of the cyst was transparent and the contents were perfectly clear. A needle was inserted through the capsule of the cyst and 28 cc. of clear, colorless fluid were obtained. The fluid contained 270 mg. per cent of protein. After the cyst had been completely evacuated the opening into its capsule was enlarged and exploration of the cyst was carried out, but no solid tumor was found. A piece of the wall was removed for microscopic examination. The patient was in excellent condition throughout the operation.

Microscopic Examination. The surgical specimen was a portion of the thin cyst wall (Fig. 1). The inner surface was formed by a single layer of epithelial cells which varied from the columnar type in some areas to the cuboidal type in others (Fig. 2). No cilia were seen on care-

FIG. 1. Photomicrograph of the wall of the cyst at low magnification. Hematoxylin and eosin.

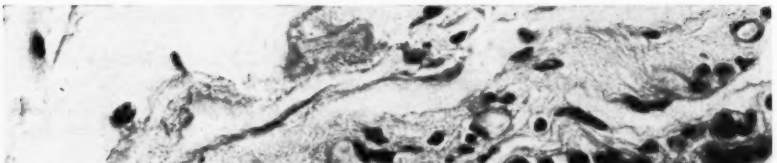

FIG. 2. Section of the wall showing a single layer of columnar and cuboidal cells lining the cyst. Hematoxylin and eosin ×440.

ful examination under the oil immersion lens. Next to the lining there was a loose stroma of supporting connective tissue. Within this stroma an occasional endothelial-lined capillary was to be seen. In certain areas of the connective tissue there were nests of cells possessing darkly stained oval and spherical nuclei.

This was judged to be a cyst of hypophyseal origin lined by non-ciliated columnar and cuboidal epithelium.

Postoperative Course. For several days the patient had a fever which did not rise above 102°F. The peripheral visual fields showed the left field to be full but there still remained a defect of the upper temporal quadrant on the right.

The patient was last seen in November 1952, 4½ years after the operation. The visual fields were unchanged. There was still a right upper temporal quadrantanopsia. The visual acuity of the right eye was 20/30 and of the left eye 20/20, and both optic discs were still pale. During the 4½ years he had remained asymptomatic demonstrating no evidence of further pituitary or hypothalamic involvement. He had no complaints and had continued his work as a contractor.

DISCUSSION

In 1920 Duffy[2] described a comparable cyst as did Frazier and Alpers[3] in 1934. However, these cysts differed from the one reported here in that the lining cells possessed cilia. At autopsy Russell[6] encountered four similar cysts: one suprasellar, one intrasellar and two dumbbell-shaped tumors with both intrasellar and suprasellar parts, the constriction being at the diaphragma sellae. With the exception of the suprasellar portions of the dumbbell-shaped cysts, which were lined with squamous epithelium, each of the cysts that she examined was lined by a single layer of ciliated epithelium. In our case no cilia could be found. As this was a surgical

specimen which was promptly fixed it seems unlikely that cilia could have been present and later disintegrated. In other respects the lining cells in this cyst seemed similar to those in the others, i.e., they were columnar epithelial cells arranged in a single layer.

One should not be surprised to find such cysts large enough to produce signs and symptoms, for the normal hypophysis may contain cysts lined by ciliated columnar, columnar and cuboidal epithelial and occasional goblet cells. These cysts are filled with colloid and appear chiefly in the pars intermedia and in the region of the remnants of Rathke's cleft.[1,4,5] Apparently the intrasellar cyst in this report represents one of these microscopic cysts which had enlarged because of the increased secretory activity of the wall of the cyst. Why these cysts rarely attain sufficient size to produce symptoms remains a mystery and invites speculation.

With the exception of Russell's suprasellar cysts, these uncommon lesions have been intrasellar and consequently are impossible to differentiate clinically from other intrasellar space-occupying lesions such as the adenomata. This case presented findings typical of a hypophyseal chromophobe adenoma—bilateral upper temporal quadrantanopsia, hypopituitarism, bilateral optic atrophy and ballooning of the sella turcica.

It is very interesting and unexpected that the patient in our report has remained symptom-free for as long as $4\frac{1}{2}$ years. In Duffy's[2] case the patient, who had been operated on by the late Dr. Walter E. Dandy, expired 9 hours postoperatively. Frazier and Alper's[3] patient was followed for 3 years postoperatively and during this time was gainfully employed and asymptomatic. His whereabouts and condition after that time are not known. Judging from our case and that reported by Frazier and Alpers it would appear that after the successful evacuation of a cyst of this sort the patient has an excellent outlook.

SUMMARY

The case of a man 41 years of age with an intrasellar cyst lined by columnar and cuboidal epithelium is reported. The symptoms and findings were suggestive of a chromophobe pituitary adenoma. At operation the cyst was evacuated. During the ensuing $4\frac{1}{2}$ years the visual fields have improved and the patient has remained asymptomatic. This case and the only similar one reported in which the patient survived operation indicate that the prognosis for life and recovery from symptoms after evacuation of this type of cyst is exceptionally good.

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RUPTURED INTERVERTEBRAL DISC

REPORT OF A CASE WITH A DEFECT IN THE ANTERIOR ANNULUS FIBROSUS

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There are 2 cases reported in the literature in which vascular complications occurred as a result of disc surgery. Linton and White[3] in 1945 reported a case of an arteriovenous fistula between the right common iliac artery and the inferior vena cava which followed operation upon a ruptured lumbosacral disc. The patient had shortness of breath, an enlarged heart and an abdominal bruit, and the diagnosis of arteriovenous fistula was made 8 months later. The vascular lesion was subsequently operated upon and was thought to have resulted from the disc surgery. Holscher[2] in 1948 reported a case of vascular injury caused by disc surgery. This patient had a ruptured intervertebral disc on the left at the 4th lumbar level. It was stated that the surgeon was very careful when he palpated the anterior annulus fibrosus with a closed pituitary rongeur. His instrument slipped with surprising ease through the interspace. After the rongeur was removed, there was a copious welling-up of dark blood. The bleeding was controlled by packing. Subsequently an abdominal bruit and thrill developed, and a diagnosis of arteriovenous aneurysm was made. Six months later corrective vascular surgery was carried out. A 3 cm. arteriovenous aneurysm was found between the right common iliac artery and vein.

The authors have learned of 8 fatalities resulting from vascular injury at the time of disc surgery. The deaths were caused by massive, intra-abdominal hemorrhage. Holscher,[2] in his report, said that he knew of 4 other unpublished instances of vascular injury resulting from disc surgery. It is our opinion that many more than 14 patients have had vascular complications attributable to intervertebral disc operations. Probably in most of the known complicated cases the operations were performed by neurological surgeons. It seems likely that the emphasis that Dandy[1] placed on removing "the entire vertebral disk" precipitated the more radical trend resulting in these complications. The case to be reported indicates that the great vessels anterior to the lumbar vertebrae could be injured as a result of abnormality of the anterior annulus fibrosus.

CASE REPORT

A 34-year-old white male oil-worker was admitted to the Houston Veterans Administration Hospital on July 22, 1950. He complained of episodes of low back pain of 2 years' duration with pain radiating down the back of the right leg into the heel. This pain was made worse by straining, coughing or lifting.

Examination. He had a positive Naffziger test, a positive straight leg raising test and a positive Lasègue sign. His right ankle jerk was absent and there was hypesthesia of the right S1 dermatome.

Roentgenograms of the lumbar spine and sacrum showed that there was no narrowing of the intervertebral spaces. The lordotic curve was normal and there was scoliosis with the

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convexity to the right. The sacro-iliac joints appeared normal. A diagnosis of herniated nucleus pulposus of the 5th lumbar disc on the right was made.

Operation. On Aug. 2, 1950, under spinal anesthesia, a midline incision was made over the lumbosacral area to expose the right lumbosacral interspace. The interlaminar space was enlarged slightly with the rongeur and then the ligamentum flavum was excised. The 1st sacral root and dural sac were retracted medially, and a large piece of loose nucleus pulposus was grasped and removed with the pituitary rongeur. When the closed pituitary rongeur was placed into the annulus defect it passed into the interior of the intervertebral disc, and then fell farther with minimal pressure and with no additional rongeuring. With the thought that a fissure through the anterior annulus fibrosus had been entered, 2 cc. of pantopaque were dropped without pressure into the posterior annulus defect.

To complete the removal of the nucleus pulposus adjacent to the defect, care was exercised not to enter the anterior fissure. After routine wound closure the usual anteroposterior and lateral roentgenograms were made to determine the position of the iodized oil. The lateral film shows best the anterior position of the pantopaque (Fig. 1) which appears to be in front of the anterior longitudinal ligament.

Course. The patient made a prompt recovery. He left the hospital on leave on Aug. 9, 1950, and was discharged 2 weeks later.

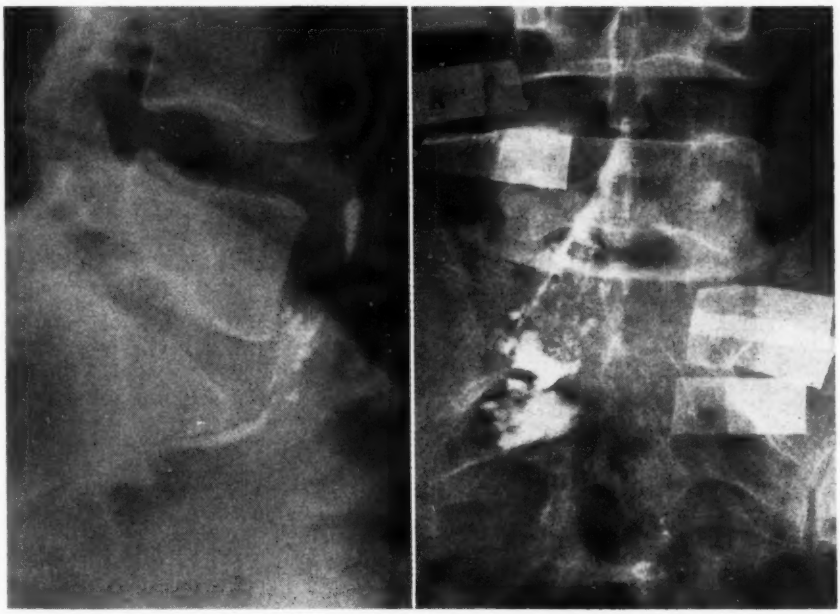

FIG. 1. Lateral and anteroposterior roentgenograms of the lumbosacral spine, showing the position of the pantopaque after it passed through the anterior annulus defect. The lateral film indicates that the oil lies in front of the anterior longitudinal ligament.

Comment. The major vessels anterior to the vertebral column have been injured in the past by excessive surgical effort. The possibility of an anterior defect in the annulus fibrosus must prompt caution in the use of sharp instruments within the intervertebral disc.

SUMMARY

1. Two patients with vascular complications following disc surgery have been reported in the literature. We are familiar with 12 other cases of vascular complications from disc surgery. Most of these terminated in death as a result of injury to the great vessels anterior to the 5th lumbar vertebra or adjacent vertebrae.

2. A case in which the presence of a fissure of the anterior annulus fibrosus was demonstrated by roentgenograms after injection of iodized oil is reported. This may occur more frequently than we realize and may be the explanation of some of the vascular injuries that have occurred even when extreme care was exercised during disc surgery.

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PROSTATIC METASTASES SIMULATING INTRACRANIAL MENINGIOMA

A CASE REPORT

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(Received for publication February 24, 1953)

The following case illustrates an unusual manifestation of carcinoma of the prostate. The radiological findings appeared pathognomonic of the hyperostosis accompanying meningioma *en plaque*, so that other features were completely overlooked. It was only after the somewhat painful surprise of the histological diagnosis that we became aware of the importance of these discrepancies.

CASE REPORT

A 71-year-old white male complained in July 1952 of progressive protrusion of the right eyeball for 4 months and numbness over the right eye. For many years he had been almost totally blind in the left eye.

Present Illness. In the latter part of March 1952 the patient had severe pain in the back of the right eye and in the right side of the face, including the forehead and jaw. About 1 week later the right eye protruded so that it touched the lens of his glasses. The pain lessened markedly after the eye protruded. On July 4, the patient first noticed numbness of the area that formerly had been the site of the pain. The protrusion of the eye was greater at this time. The only objective findings were slight residual numbness over the right eye and protrusion of the eyeball. There had been an indefinite loss in weight.

Examination, July 17, 1952. The sense of smell was normal. There was a nonpulsating exophthalmos of the right eye, of severe degree (Fig. 1). All ocular movements were markedly impaired but there was no evidence of paralysis of the individual muscles of the eye. Corneal sensation was normal. There was papilledema of 2–3 D. in the right eye and engorgement of the veins, with flame-shaped hemorrhages. Vision in the right eye was reduced to some light perception. There was amblyopia ex anopsia in the left eye. Moderate anesthesia and analgesia were present over the distribution of the 1st and 2nd divisions of the right 5th nerve. The 3rd

division was intact. There seemed to be an enlarged lymph node in front of the right mastoid process and there was a suggestion of thickening of the skull deep to the temporal muscle in the anterior part of the right temporal fossa. Rhinological studies were negative.

Roentgenograms of the skull showed a considerable hyperostosis involving the entire sphenoid wing, the zygomaticotemporal process and a fair portion of the floor of the middle cranial fossa surrounding and posterior to the sella turcica (Figs. 2 and 3). Both bony borders of the superior orbital fissure were involved and the fissure itself was somewhat narrowed. The optic foramen was completely surrounded by hyperostotic bone, but appeared to be relatively nôrmal in size (Fig. 4). There was nothing in the radiographs to suggest increased intracranial pressure. The pineal body was not visualized. Impression: Meningioma of the sphenoid wing, resulting in considerable hyperostosis.

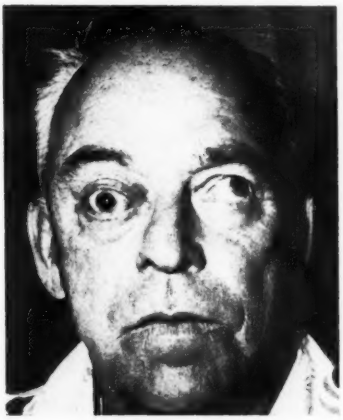

FIG. 1. Photograph of patient showing proptosis of the right eye. He had been almost totally blind in the left eye for many years.

Clinical Diagnosis. The original clinical diagnosis was malignant tumor of the nasopharynx with extension along the base of the skull. However, after the radiographs were seen it was changed to meningioma *en plaque* arising in the right pterion. It was pointed out, however, that it is rather uncommon to have this condition in an elderly male patient and, furthermore, that the history of such rapidly progressive exophthalmos was unusual. In order to exclude the presence of a global intracranial extension, cerebral angiography was recommended.

Angiography, July 17, 1952. A right carotid angiogram showed good filling of the branches of the anterior, middle, and posterior cerebral arteries.

FIGS. 2 and 3. (*Left*) Frontal projection of skull showing hyperostosis of the bones about the orbit and sphenoid wings. (*Right*) Lateral projection demonstrating similar changes in the floor of the anterior cranial fossa.

FIG. 4. Oblique view showing similar changes. Note involvement of the zygomaticotemporal process. In this view, the optic foramen is seen to be normal in size.

There was only minimal contralateral filling demonstrated. There appeared to be some elevation of the first and second portions of the right internal carotid artery and of its main branches in their first portions. However, the second portion of the middle cerebral artery and the second portion of the anterior cerebral artery both appeared to be minimally depressed. These observations suggested that in addition to a tumor mass below the bifurcation, there was some extension above the bifurcation. There was no definite evidence of displacement of the posterior cerebral artery.

Clinically and roentgenologically, therefore, we were presented with a 71-year-old patient who appeared to have a rather typical meningioma *en plaque* originating in the right pterion. Ordinarily surgery in such an individual would not be indicated since full removal of involved bone would be formidable. In this particular case, however, it was decided that decompression of the right optic nerve might prevent further loss of vision in the right eye of a man now almost completely blind in the other eye.

Operation, July 23, 1952. According to a technique previously published[3] a bone flap measuring 3×3 cm. was removed over the right temporal fossa and sphenoid bone. The pole of the temporal lobe and the posterior part of the frontal lobe were exposed. The inferior aspect of the temporal muscle next to the bone appeared invaded with tumor. The bone of the temporal process and greater wing of the sphenoid was greatly thickened and was removed. Finally a piece of bone was removed which appeared to be the anterior clinoid process. In the depths the optic nerve and the anterior cerebral artery were clearly visualized. The optic nerve apparently had been grooved just about where the clinoid process had been. The superior and lateral aspects of the orbit were removed and the periorbital fascia was incised and found to be thickened to at least 2–3 mm. The appearance was that of tumor tissue similar to a meningioma. The dura mater itself was thickened in one place but no definite plaque was

found. The thickened area of dura mater was removed. The optic nerve seemed now to be completely free of pressure. The wound was closed by using the muscle flap to cover the defect in the dura mater. The bone was not replaced.

Histological Diagnosis. Four biopsy specimens were examined, taken from the following tissues: (1) periosteum and temporal muscle, (2) bone, (3) thickened dura mater, and (4) periorbital tissue. In all the specimens, nests of anaplastic and apparently metastatic carcinoma were found.

Postoperative Course. The patient withstood the procedure well. A radiological survey of the bones was now made which showed spotty areas of increased density scattered through the bodies of the lumbar vertebrae and both halves of the pelvis as well as the upper visualized portions of both femurs. These areas of increased density were spherical, of various sizes, and characteristic of the osteoblastic metastases of carcinoma of the prostate. On rectal examination the right lobe of the prostate was found to be hard in one area, suggesting a carcinoma. The patient was discharged on Aug. 2, 1952.

Subsequently he failed progressively and by Oct. 23, 1952 his condition was such that he was unable to leave his home. His vision had not improved following the operation, and he had been unable to return for re-examination. He expired early in November, 1952. At autopsy the diagnosis of carcinoma of the prostate was confirmed.

DISCUSSION

The general medical and radiological literature of the past 20 years fails to reveal other similar cases. A case of prostatic carcinoma was reported by Bonnet and Colrat[1] in which exophthalmos, loss of visual acuity, and opacity in the orbit are described. No bony involvement is mentioned and there appears to have been no difficulty in differentiation from a meningioma *en plaque.* No roentgenograms were reproduced. No instance in which there was hyperostosis associated with metastatic tumors in the meninges is included in the Brain Tumor Registry.[4] It has been generally conceded that the syndrome of hyperostosis in the floor of the anterior cranial fossa, together with a gradual loss of visual acuity and exophthalmos, is practically pathognomonic of meningioma *en plaque.* In this case several important features were not given appropriate emphasis; the rapidity of the loss of vision and exophthalmos in the right eye, the patient's age and sex, and the hyperostotic involvement of the zygomaticotemporal process. Ordinarily meningioma *en plaque* is a disease of females in the fourth decade characterized by exophthalmos, gradual loss of vision, and distinctive bony changes.[2] Doubt was raised first on failure to find the expected dural plaque at operation. In this case, discrepancies should have led us to re-evaluate our position and to consider particularly metastatic disease.

We believe that in cases in which decompression of an optic nerve might restore or preserve vision, surgery is warranted.

SUMMARY

A case report is presented of a 71-year-old white male suffering from carcinoma of the prostate with metastases to the skull, superficially simulating a meningioma *en plaque.*

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CONGENITAL BERRY ANEURYSM OF THE POSTERIOR FOSSA

CASE REPORT WITH SUCCESSFUL OPERATIVE EXCISION

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(Received for publication March 23, 1953)

The intracranial operative attack upon congenital berry aneurysms has been successfully accomplished on numerous patients. However, most of these successes occurred in cases of supratentorial aneurysms. In an excellent monograph, Dandy[1] reported that he encountered a large saccular posterior fossa aneurysm in 2 cases when operating for a suspected posterior fossa tumor. Though these patients died following surgery, he predicted that, in certain cases, posterior fossa aneurysm would certainly be cured surgically. Schwartz[2] was the first to report an operative cure of a posterior fossa saccular aneurysm in a 28-year-old woman with a cerebellopontine angle syndrome. Before his report appeared, we had been fortunate in effecting the operative cure of a saccular posterior fossa aneurysm of the left posterior inferior cerebellar artery in a patient with subarachnoid hemorrhage in whom the site of the ruptured aneurysm was not suspected until hydrocephalus had been demonstrated by ventriculography. Exploration of the posterior fossa and excision of the aneurysm resulted in an apparent cure. We are not aware of any other cases in which there was successful outcome following surgery. There probably have been some unreported cases, for vertebral angiography has been more common since the report of Sugar *et al.*[3] This diagnostic aid will certainly lead to the discovery and cure of many more posterior fossa aneurysms.

CASE REPORT

A 24-year-old white, male sergeant was admitted to the Walter Reed Army Hospital on Dec. 16, 1946. The only available history, the accuracy of which has never been fully confirmed, is that the patient had been in a fight at approximately 0100 hours on December 16. He was transferred through medical channels, reaching Walter Reed at approximately 1500 hours, Dec. 16, 1946.

Examination. He was stuporous, irritable, and disoriented as to time, place, and person. There was bilateral 6th nerve paresis, greater on the right. The pupils were small and equal. Funduscopic examination revealed multiple subhyaloid hemorrhages bilaterally, more marked on the right. The right optic disc was somewhat pale. The other cranial nerves were intact. The tendon reflexes could not be obtained. There was a left positive Babinski sign. The neck was markedly stiff and a positive Kernig's sign could be demonstrated. A lumbar puncture was performed revealing an initial pressure of 400 mm. of water with grossly bloody fluid. Blood and urine were normal.

A diagnosis of subarachnoid hemorrhage caused by rupture of a congenital aneurysm was made.

Course. The patient was observed on the Neurological Service and by the Medical Service. He maintained an elevated blood pressure ranging from 140/105 to as high as 170/130. His temperature varied, ranging from as high as 102.8° at some times to as low as 98.6°. His respiratory rate was from 20 to 30 per minute. His pulse rate varied; it was as high as 160, but usually remained 120 per minute.

On Jan. 14, 1947, bilateral temporal and bilateral occipital burr holes were made; there was no evidence of subdural hematoma. The patient did not improve and on Jan. 20, 1947 ventriculography was performed through the previously made burr holes. It revealed symmet-

ical dilatation of the lateral ventricles with an enlarged 3rd ventricle and aqueduct; there was no displacement of this portion of the ventricular system. However, the portion of the 4th ventricle that could be visualized was thought to be shifted to the right. The hydrocephalus was interpreted as being caused by either a ruptured congenital aneurysm in the posterior fossa, or by a subarachnoid block secondary to the subarachnoid hemorrhage.

Operation. On Jan. 23, 1947, when the patient was moribund, a posterior fossa exploration was carried out with the usual transverse incision. An aneurysm, 2 cm. by 1 cm., was disclosed lying beneath and anterior to the left cerebellar tonsil. It arose from the posterior inferior cerebellar artery. There was evidence of recent rupture of the aneurysm at its distal end. In attempting to mobilize the neck of the aneurysm, bleeding was encountered and accordingly silver clips were placed on the vessel proximally and distally to trap the aneurysm. The aneurysm was then excised with a segment of the vessel from which it arose (Fig. 1).

FIG. 1. Excised collapsed aneurysm after escape of blood.

Course. Immediately postoperatively the patient maintained the hypertension previously noted and also had a tachycardia ranging about 140. His respiratory rate ranged between 24 and 30 and his temperature remained as high as 104–104.4°. The most annoying problem during his postoperative phase was persistent hiccoughs, but fortunately these disappeared.

By Feb. 4, 1947 the patient was able to be up in a chair. He had bilateral ataxia, marked, of both arms and great weakness of both legs. The latter apparently was the result of his protracted illness as it was not possible to detect any true neurological changes, either motor or sensory. Complete neurological evaluation was difficult because of his confusion and poor cooperation.

By February 24 the blood pressure was within normal range, 110/70, and the temperature was normal, as were the pulse and respirations.

The patient was given intensive physical therapy with particular care to ambulation. At first, difficulty with walking was observed. Also the patient remained disoriented with a very short memory span; however, by May 31 he was able to go on a convalescent furlough with a practically normal gait and only mild euphoria. After returning from his furlough, psychometric examination was carried out on Aug. 5, 1947. His total I.Q. was 97, a marked improvement over his previous I.Q. of 78 on May 13, 1947. On discharge he showed mild euphoria and a slight degree of ataxia in the left arm.

It has not been feasible to re-examine this patient since his discharge from the military service. However, he reported by telephone in October 1952 that he had recently completed a two-year training course as a mechanic and was now working as a mechanic. He stated that he was asymptomatic except for slight incoordination in the use of the left hand.

SUMMARY

A case is reported of a saccular aneurysm of the left posterior inferior cerebellar artery in a man with subarachnoid hemorrhage. Its location was first suspected when ventriculography showed a hydrocephalus with a shift of the 4th ventricle to the right. The aneurysm was trapped and excised, with successful outcome.

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PNEUMOCISTERNOGRAPHY OF THE POSTERIOR FOSSA

PRELIMINARY REPORT OF A NEW TECHNIQUE

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(Received for publication February 18, 1953)

We have recently developed a new method for the visualization of the ventricular and cisternal cavities of the posterior fossa, by injection of air into the lumbar subarachnoid space. The main feature of this technique is the fact that the gas does not reach the intracranial cavity while it is being injected, but collects first in the lower part of the subarachnoid space and then ascends to the region of the foramen magnum in the quantity and with the speed and direction desired by the operator. This "control" of the air is obtained by tilting the radiological table on which the patient is lying. In this preliminary report the technical aspects of the procedure will be illustrated. A full consideration of indications, results and comparative advantages will be deferred until a later date.

TECHNIQUE

The patient is placed on his side and a lumbar puncture needle is inserted in the 3rd and 4th lumbar interspace. Manometric readings are taken (Fig. 1) and then the table is tilted 2 or 3 degrees in order to have the head-level slightly lower than the foot-level (Fig. 2). Eight cc. of cerebrospinal fluid are withdrawn and 10 cc. of air are injected, and this is repeated until

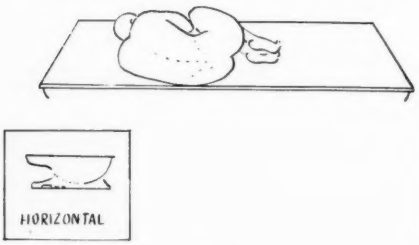

FIG. 1

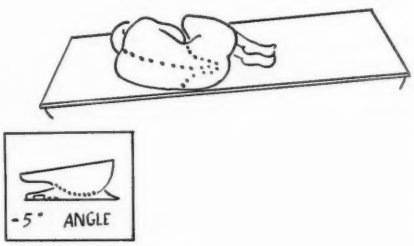

FIG. 2

30 cc. of fluid have been withdrawn and 40 cc. of air injected. To avoid a possible escape of air, following the last injection, the needle is removed attached to the syringe. The patient is rolled over on his abdomen, a small sandbag is put beneath the forehead, and the head is placed so that the orbitomeatal line is perpendicular to the plane of the table. The patient's head is still at a lower level than the rest of the body (Fig. 3). The patient is now instructed not to move his head in any direction. A foot-rest prevents him from slipping downward. A 10×12 inch cassette, screened by a Lysholm grid, is placed against the patient's head. The head of the table is tilted slowly 25° upward (Fig. 4), and three lateral views, at about one minute intervals, are taken with the tube centered on the mastoid. Fourth and fifth lateral views are taken with the table tilted an additional 10°. The head is slightly and fully dorsi

* Smith-Mundt-Fulbright Fellow from University of Palermo, Palermo, Italy.

flexed for the fourth and fifth exposure, respectively (Figs. 5 and 6). Finally the table is brought back to the horizontal position and two postero-anterior views are taken with the tube inclined 10° and 30°, with the normal ray aiming at about 2 cm. below the protuberantia occipitalis externa. When visualization of the whole ventricular system is required, routine views can be taken, as the amount of air is usually enough to give a good outline. In those cases in which there is increased intracranial pressure only 25 cc. of air are injected, 5 cc. at a time,

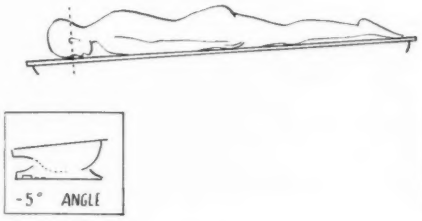

FIG. 3

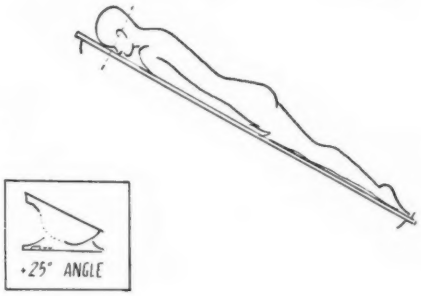

FIG. 4

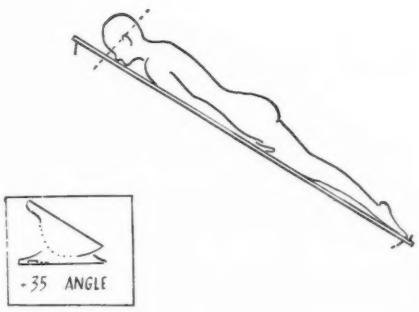

FIG. 5

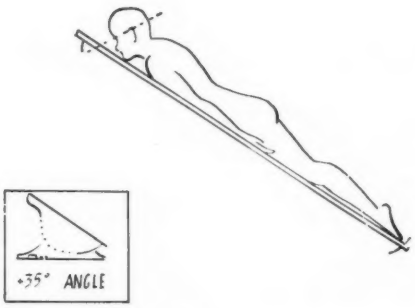

FIG. 6

and a corresponding amount of cerebrospinal fluid is withdrawn. At times following the injection of air no outflow of fluid or a mixture of fluid and air will occur and it will be necessary to bring the table to the horizontal position, or a few degrees more to facilitate fluid-air exchange. In this respect a needle inserted in the 3rd lumbar interspace will represent an advantage. The air starts entering the intracranial cavity when the head of the table is elevated about 10°.

RESULTS

The cisterna magna, the 4th ventricle, the aqueduct, and the posterior part of the 3rd ventricle are usually visualized in the first three lateral views (Fig. 7). The cisterna venae magnae Galeni is also frequently outlined as well as the vermis (Fig. 8), or at times the entire cerebellum (Fig. 9).

In the fourth and fifth lateral views, air is seen in the occipital horns and the pontine, interpeduncular and ambiens cisterns (Fig. 10).

FIG. 7

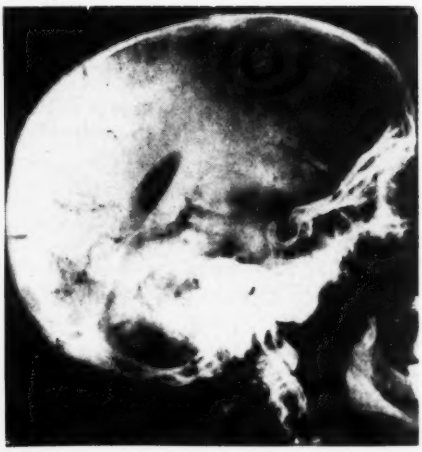

FIG. 8

FIG. 9

FIG. 10

COMMENT

We consider this technique advantageous because: (a) The exchange of fluid-gas is done with the patient in horizontal position which makes the procedure safer and less disturbing, particularly in cases of increased intracranial pressure. (b) No special radiological equipment is required, only a standard tilting table being necessary. (c) The amount and the movement of the gas are fully "controlled."

Thanks are gratefully expressed to Dr. Francis J. O'Neill, Director of the Central Islip State Hospital, and to Dr. Thomas I. Hoen, Director of Neurosurgery, for assistance and advice in the development of this technique.

A SIMPLE, GENTLE, SELF-RETAINING BRAIN RETRACTOR

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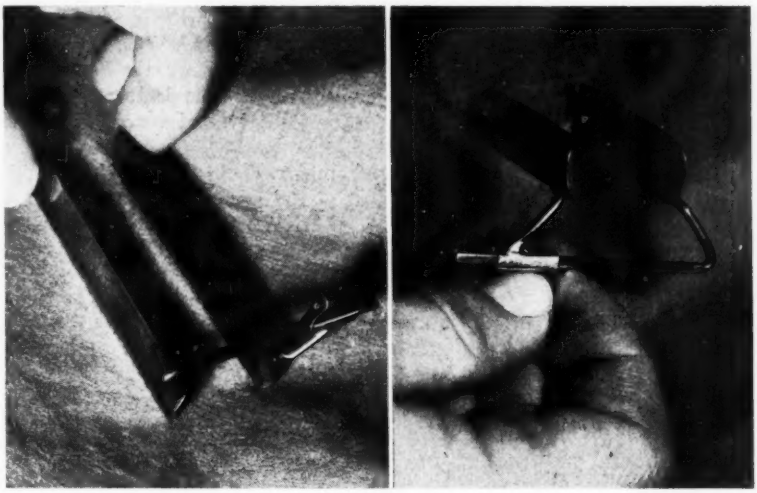
(Received for publication February 24, 1953)

A simple, handy, gentle, self-retaining brain retractor has been in use in the author's hands for the last year. Many times during intracranial procedures, brain spatulas or brain retractors are held by the surgeon and his assistants in order to maintain adequate exposure of deep-seated tumors. This is particularly true during transventricular procedures, separation of the cerebellar hemispheres during fourth ventricle explorations, and during the removal of a mural nodule in cystic gliomas. This instrument was used also in a series of transventricular psychosurgical procedures during which micro-electrodes were inserted into the septum pellucidum for the performance of electrical stimulation of these and other subcortical nuclei (to be published).



FIGS. 1 and 2

The instrument* is a modification of the Patton self-retaining nasal speculum (Figs. 1 and 2). It is exceedingly gentle on the retracted brain substance and it stays open to the desired diameter with the mere pressure of the cerebral tissue on its blades. It has the dual advantage that it does not move and does not dig into the brain tissue as so frequently happens when an assistant is holding a brain retractor for a prolonged period of time.

* This instrument can be obtained at the Coshow Specialty Shop, 1801 St. Claude Ave., New Orleans 16, La.

NOTICES

AMERICAN NEUROLOGICAL ASSOCIATION

At the 78th annual meeting of the American Neurological Association held in Atlantic City, New Jersey from June 15–17, 1953, the following officers were elected for the year 1953–1954: President, Dr. Roland P. Mackay; President-Elect, Dr. Percival Bailey; First Vice-President, Dr. William G. Lennox; Second Vice-President, Dr. Russell Meyers; Secretary-Treasurer, Dr. H. Houston Merritt; Assistant Secretary, Dr. Charles Rupp.

CONGRESS OF NEUROLOGICAL SURGEONS

The third annual meeting of the Congress of Neurological Surgeons will be held November 12–14, 1953, in the Roosevelt Hotel, New Orleans, Louisiana. Members of the medical profession are welcome to attend.

Sir Geoffrey Jefferson, Consulting Neurosurgeon of the Manchester Royal Infirmary, will give the opening address on Thursday at 11:00 A.M. and will speak again on Friday at 11:00 A.M. and on Saturday at 11:00 A.M. The following panel discussions will be held: "The anatomy and physiology of the frontal lobes" (Robert Heath, Moderator, Stanley Cobb, Harold Himwich, Gerhardt von Bonin, and A. Earl Walker). "Psychosurgery: indications and sequelae" (A. Earl Walker, Moderator, Stanley Cobb, Gerhardt von Bonin, and J. Lawrence Pool). "The use of fluids and electrolytes in the management of the neurological patient" (Hyman Mayerson, Moderator, Harold Himwich, Alton Ochsner, J. Lawrence Pool, A. Earl Walker, and Walter Wilde).

Further information may be obtained from Dr. Henry M. Suckle, 414 Tenney Building, Madison 3, Wisconsin.

SUBDURAL HEMATOMA OCCURRING IN SURGICALLY TREATED HYDROCEPHALIC CHILDREN

WITH A NOTE ON A METHOD OF HANDLING PERSISTENT ACCUMULATIONS

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WHILE in recent years subarachnoid-peritoneal anastomoses have been performed, the majority of patients with congenital hydrocephalus have been treated in this clinic by choroid plexectomy. Our technique for the operation has been reported in detail elsewhere.[2] In order to maintain the integrity of the cranial vault when cerebrospinal fluid is withdrawn from the ventricle, a plaster cast is applied preoperatively in the form of a ring encompassing the forehead and occiput. Following excision or coagulation of the plexus, Ringer's solution is instilled into the ventricle in sufficient quantity to prevent the brain, which has been supported during the course of the operation with retractors, from collapsing. If the head is so positioned that the opening in the skull is uppermost, the fluid can be replaced almost completely. However, it is very difficult to expand the brain so as entirely to obliterate the subdural space, and some separation between cortex and dura mater may persist. This space is traversed by a variable number of stretched veins, which may conceivably rupture and produce a hematoma.

Surgical procedures for the treatment of hydrocephalus other than choroid plexectomy, designed to reduce intracranial pressure by shunting cerebrospinal fluid into extraneural channels, pose a similar threat. A sudden diminution of pressure within the ventricles resulting from withdrawal of cerebrospinal fluid may also possibly bring about a collapse of the brain which, in turn, may lead to rupture of a vein and bleeding into the subdural space.

Though mentioned in a paper by Voris,[3] to our knowledge there is only Anderson's[1] report in the literature dealing primarily with subdural hematoma as a complication of the surgical treatment of hydrocephalus. The latter has described 3 such cases occurring in a group of 24 treated infants. In all 3 symptoms referable to the hematoma became manifest early in the postoperative course. The hematoma developed after a spino-ureteral anastomosis in 1 case, following choroid plexectomy in a 2nd and subsequent to the removal of an intraventricular cyst containing choroid plexus in a 3rd patient.

In view of the paucity of information available on this subject, we are reporting 3 additional cases. Incidentally a method of dealing with chronic recurrent subdural accumulations of fluid in a head much enlarged by pre-

ceding hydrocephalus will be briefly described. Our experience is based on 85 surgically treated hydrocephalic children on whom 134 operations were performed.

CASE REPORTS

Case 1. B.I.H. #245005. R.B., hydrocephalic male. Bilateral choroid plexectomy at 3½ months. After 2 years, trauma to head; left subdural hematoma disclosed. Reaccumulation of subdural fluid after classical treatment. Control after subdural-cisternal anastomosis.

History. A 3½-month-old male infant was referred to this clinic on Feb. 23, 1948 because of excessive enlargement of the head. He was a first-born child delivered by forceps after a normal full-term pregnancy. A scalp laceration in the left frontal region was incurred at the time of delivery.

Examination. The pertinent findings were limited to the head. It measured 48.5 cm. in circumference. The anterior fontanelle was dilated (6×6 cm.) and bulged moderately.

Course. Subdural punctures failed to disclose the presence of a hematoma. Accordingly a ventricular estimation was performed using a small quantity of gas (40 cc.). An extreme degree of ventricular dilatation was revealed.

1st and 2nd Operations. Under local anesthesia a small right parietotemporo-occipital flap was reflected and a choroid plexectomy was performed on Feb. 26, 1948. Twelve days later a similar procedure was done on the left side.

Course. The child withstood the operations well and at the time of discharge from the hospital on Mar. 16, 1948, his head circumference measured 49 cm.

For about 1½ years, he appeared to be making satisfactory progress. His fontanelle closed, he began to walk and his mental development was seemingly reasonably good. At the age of 8 months an internal squint of the left eye became manifest. Some enlargement of the head continued to take place, its circumference in September, 1949 being 53.5 cm.

In January, 1950, following a fall, the child became irritable, drowsy and vomited occasionally. In addition to these symptoms, episodes occurred during which he would suddenly retract his head.

Readmission, Jan. 11, 1950. Examination at this time disclosed no gross neurologic abnormalities. Lumbar puncture revealed xanthochromic CSF containing 1822 mg. per cent total protein. EEG indicated an absence of all electrical activity over the left hemisphere. The presence of a subdural hematoma was suspected.

3rd Operation. Bilateral trephination was performed on Jan. 15, 1950. A large subdural clot overlying the left cerebrum was evacuated.

Course. Considerable improvement followed.

4th Operation. Removal of the inner membrane of the subdural hematoma was subsequently accomplished by means of a craniotomy. Fluid similar in character to that present in the subdural space was found within the ventricle, leading to the conclusion that the subdural space and ventricle communicated through the opening of the cortex originally created for the purpose of gaining access to the choroid plexus.

Course. Right-sided seizures developed, but otherwise the child's course was not remarkable. Except for occasional vomiting, his condition during the next 8 months was reasonably satisfactory. An EEG in March, 1951 was reported to be within normal limits.

In June, 1951 hospitalization was again necessary because of recurrence of seizures. Again no definite neurologic abnormalities were detected. His behavior, however, was such as to indicate that in all probability he was mentally retarded. The circumference of his head measured 54 cm. A pneumoencephalogram attempted by way of the lumbar route was unsuccessful. The CSF was xanthochromic and contained 168 mg. per cent total protein. A subdural puncture on the left side yielded brown fluid demonstrating a persistence of the hematoma. The subdural space on the right side was inspected through a trephine opening and found free of clot.

5th Operation. On July 10, 1951, utilizing the principle of the Torkildsen procedure, a connection was established by means of a polyethylene tube between the subdural space on the left side and the cisterna magna for the purpose of draining the chronic subdural accumulation.

Subsequent Course. During the time that has elapsed since the last operation, the circumference of the child's head has increased only 1 cm. At varying times he has vomited and occasionally complained of headache. Since August, 1951 he has been free of major seizures. His intelligence has continued below par.

Case 2. B.I.H. #237853. M.G., 3-year-old girl, with progressively enlarging head since birth. Normal intelligence. Head circumference 60 cm. Separation of sutures. Subarachnoid-peritoneal anastomosis. After 1 month, bilateral subdural hematoma. Failure of classical operations. Control by subdural-cisternal anastomosis.

History. A female child, aged 3, was first seen in October, 1950 because of an unusually large head, present since birth. As the child grew older, the head had continued to enlarge disproportionate to the size of her body. Except for some unsteadiness of gait, her mental and physical development were said to have been normal.

Examination. The patient appeared to be a remarkably bright child, active, friendly and talkative. Her head was considerably enlarged, measuring 60 cm. in circumference. The anterior fontanelle was still partly open. On percussion a cracked pot sound was audible. She exhibited a slightly spastic gait with a tendency towards adduction of the lower extremities and inversion of the feet. Roentgenograms of the skull disclosed separation of the sutures.

Course. Communication between the lateral ventricle and lumbar subarachnoid space was demonstrated by means of the tilt test.

1st Operation. On Nov. 7, 1950 a lumbar laminectomy was performed and a subarachnoid-peritoneal anastomosis was established by means of a polyethylene tube.

Course. An electrical amplitude diminution, which had been noted preoperatively over the right frontal and parietal and the left temporal regions, was still discernible following this procedure. However, whereas 2–5 per sec. waves were observed posteriorly in the record prior to operation, their frequency had increased to 4–8 per sec. postoperatively.

About a month after operation seizures involving the left half of the body developed, which resulted in a transient left hemiparesis. Following the administration of anticonvulsant medication, the convulsions did not recur.

In January, 1951 she began to complain of headache and pain in the neck and soon thereafter vomited and became drowsy. EEG now revealed a severe depression of amplitude over the entire right side, and a focus of slow activity in the occipital and temporal regions on the left side. The existence of a subdural hematoma was considered.

2nd Operation. On Jan. 30, 1951 bilateral trephination was performed. Hemato-

mas were disclosed on both sides, the one on the left being devoid of a membrane. The ventricle on the right side contained yellow fluid similar in appearance to that present in the subdural space.

Course. Following evacuation of the hematomas, the child did not improve. She frequently complained of pain in the neck, vomited and often became exceedingly drowsy. Relief usually followed withdrawal of fluid from one or the other subdural spaces.

3rd Operation. On Feb. 20, 1951 a right lateral craniotomy was performed under local anesthesia. The subdural space was emptied and the inner membrane was largely removed. The brain, however, failed to expand so that the size of the subdural cavity remained unaltered.

Course. The child recovered from the operation but her condition thereafter deteriorated rapidly. She was comatose on the 3rd postoperative day, and continuous drainage of the subdural space by catheter was instituted for 3 days. Thereafter her condition improved and she continued to gain ground.

Early in April, 1951 her symptoms returned. She again experienced pain in the neck, vomited and became irritable and drowsy. Examination revealed a bulging bone flap and bilateral extensor plantar responses. To afford her relief, it was necessary to withdraw fluid from the subdural space on one or the other side by aspiration at frequent intervals.

4th Operation. On June 3, 1951, for the purpose of allowing the subdural fluid to drain into the cisterna magna, a communication was established on the right side by means of a polyethylene tube. Over the left cerebrum, no subdural fluid was encountered, but a needle inserted through the cortex yielded yellow fluid, presumably from the ventricle. A pathway from this ventricle into the cisterna magna was created, utilizing a rubber catheter. Inspection of the region of the foramen magnum disclosed no evidence of an Arnold-Chiari malformation.

Subsequent Course. Since the last operation the child has remained remarkably well. There have occurred no manifestations of increased intracranial pressure and the circumference of the head has remained unchanged (60 cm.).

Case 3. B.I.H. #260068. P.A.M., hydrocephalic female. Right and left choroid plexectomy at 4 and 10 weeks of age. Well for 3 years. Then fall from bed and in 2 months, right subdural hematoma. Failure of classical operations. Control of subdural fluid by subdural-cisternal anastomosis. Continued symptoms because of block of foramen of Monro, probably by tumor.

History. A female infant aged 4 weeks was referred to this clinic on Jan. 12, 1949 from another hospital where a diagnosis of hydrocephalus had been established. The baby, the first-born child of her parents, was delivered by forceps following a normal pregnancy and after an otherwise uncomplicated labor. Soon after birth she began to vomit and it was noted that her head appeared unusually large. Its circumference measured 38 cm. Bilateral subdural punctures disclosed no hematoma. Ventriculography revealed dilated ventricles.

Examination. The findings of note involved the head. It was enlarged, measuring 42 cm. in circumference. The anterior fontanelle was considerably dilated (8×8 cm.), bulging and tense. There were no other abnormalities.

1st Operation. On Jan. 17, 1949 a right parietotemporo-occipital flap was made under local anesthesia and a choroid plexectomy was performed.

Course. The child tolerated the operation well and was kept under observation

thereafter for a period of about 6 weeks. Continued enlargement of the head necessitated further treatment.

2nd Operation. On March 11, 1949, choroid plexectomy was carried out on the left side.

Course. Following the 2nd operation, the child's condition was extremely satisfactory and continued so for a little over 3 years. Thus, in March, 1950 at the age of 15 months, she was able to walk and to say a few words. Her head circumference measured 53 cm. and the anterior fontanelle, though still open, was markedly scaphoid.

In April, 1951, and again a year later, her parents reported excellent progress, physically and mentally.

Some time about March, 1952 the child fell from her bed and struck her head. She was not rendered unconscious and examination disclosed no evidence of any serious injury. About 2 months later, she became listless and began to vomit and to complain of headache. When seen in August, 1952 she appeared apathetic and drowsy and examination revealed low-grade papilledema. X-rays of the skull showed separation of the sutures, thus confirming the presence of increased intracranial pressure. EEG disclosed a large amount of diffuse high voltage delta activity on the left side, as well as an asymmetry of amplitude, waves of low voltage being consistently recorded on the right.

3rd and 4th Operations. On Aug. 6, 1952, bilateral trephination was performed and the presence of a subdural hematoma was demonstrated on the right side. Two days later the clot and membranes were removed by means of a craniotomy. It was observed that the hematoma extended practically over the entire convexity of the right cerebrum.

Course. The child improved considerably immediately following the evacuation of the subdural hematoma, and the papilledema disappeared. However, about a month after operation she again became lethargic and a left hemiparesis rapidly developed. Aspiration through the right burr hole yielded dark brown fluid at a depth of about 3 cm. Roentgenograms taken after gas had been injected into this cavity made it appear that the fluid was contained within the ventricle itself.

5th Operation. On Sept. 17, 1952, a Torkildsen operation was performed, fluid from the right lateral ventricle being led into the cisterna magna through a polyethylene tube. The absence of a recurrent subdural hematoma was confirmed and the presence of clear, colorless CSF within the cisterna magna was observed. The existence of a lesion obstructing the right lateral ventricle thus appeared to have been demonstrated.

Course. Despite this procedure, however, the child's condition did not materially improve. The bone flap began to bulge and repeated lumbar punctures were necessary to lower the intracranial pressure.

6th Operation. On the assumption that absorption of CSF was defective, a subarachnoid-peritoneal anastomosis was accomplished on Oct. 14, 1952.

Course. Still the course of events was not favorably influenced. Ventriculography was performed which revealed dilated lateral ventricles blocked at the foramen of Monro.

7th Operation. On Nov. 5, 1952, through a transfrontal craniotomy, the right lateral ventricle was entered and the region of the foramen was explored. It was completely occluded seemingly by neoplastic tissue.

Subsequent Course. Following operation the child became alert, but continued to

remain hemiplegic. A course of radiation therapy was administered. To date her condition remains unchanged.

COMMENT

The relatively long interval of time that elapsed between the operations for hydrocephalus and the development of symptoms attributable to the subdural hematoma in Cases 1 and 3 may raise some doubt as to a direct causal relationship. Moreover, there was a history of trauma during the interim in each case, and the possibility that the hematomas were attributable to the trauma cannot be entirely ignored. Finally in our Case 2, as well as in 1 of Anderson's cases, a subdural hematoma developed after anastomotic procedures, without preceding craniotomy. We are inclined to believe, however, that the original operative procedures in all probability were indirectly, if not directly, the responsible factors in that they all had in common the sudden release of increased intracranial pressure in the presence of enlarged heads containing distended cerebral ventricles. It is well known that subdural hematomas may make their presence manifest after varying periods of time and, in the case of a hydrocephalic child, because of the increased capacity of the skull, this interval might be lengthened considerably. It is assumed, of course, that the hydrocephalus has been arrested since otherwise the patients would not have been asymptomatic in the intervals. It is entirely conceivable that under such circumstances a subdural hematoma might expand to a considerable size before giving rise to clinical symptoms and that the time element would be correspondingly prolonged.

The symptoms produced by the subdural hematoma in the 3 cases reported were essentially those of increased intracranial pressure. Focal seizures developed in one patient and papilledema was observed in another.

In Anderson's cases symptoms referable to the hematoma appeared much sooner following operation. Evidence of recurrent intracranial hypertension was manifested by a bulging fontanelle 10 days after a spino-ureteral anastomosis in 1 case and 3 days following removal of an intraventricular cyst in another; in the 3rd case the hematoma was accidentally encountered at a 2nd-stage operation 6 weeks after choroid plexectomy on the opposite side.

In arriving at a diagnosis various etiologic possibilities were considered. The background of hydrocephalus invariably raised the question of reactivation of the original process. Such conditions as the Arnold-Chiari malformation and aqueduct stenosis were given consideration in the differential diagnosis. The evidence that in all 3 instances suggested the possibility of a subdural hematoma was the EEG pattern. A diminution in amplitude of electrical activity over the involved hemisphere was demonstrated in each case. In the single patient with bilateral subdural hematomas the amplitude depression was recorded on the side of the more extensive lesion, while a slow wave focus was present over the opposite hemisphere. The usefulness of the EEG in providing a clue to the diagnosis in these cases cannot be overemphasized.

Difficulties were encountered in the treatment of this condition. Therapy was instituted in accordance with the plan usually adopted in infants and children. A craniotomy was eventually performed in each case, the hematoma was evacuated and the inner and outer membranes were largely excised. Persistent recurrence of the subdural accumulation in 2 patients presented a desperate therapeutic problem. Some means of providing constant internal drainage had to be devised so as to eliminate the subdural pocket of fluid, or at least annul its compressive effect. Utilizing the principle of the Torkildsen operation, the contents of the subdural space were permitted to empty into the cisterna magna through a polyethylene tube. Judging from the clinical response, this method of treatment appears to have been successful.

To minimize the likelihood of occurrence of a subdural hematoma following the surgical treatment of hydrocephalus, Anderson advised that, insofar as possible, precipitous lowering of intraventricular pressure be avoided. While undoubtedly desirable, this is not altogether feasible. A shunting operation, such as a subarachnoid-ureteral or subarachnoid-peritoneal anastomosis, would appear to be preferable to choroid plexectomy in this respect, but, judging from Anderson's as well as our own experience, obviously offers no guarantee against postoperative bleeding. Anderson further suggested that following operation the patient be kept horizontal or in the Trendelenburg position and that an adequate fluid intake be maintained.

SUMMARY

Subdural hematoma may occur as a delayed complication of the surgical treatment of congenital hydrocephalus. Three such cases are described. The importance of electroencephalography as a diagnostic aid is emphasized. Chronic recurrent subdural hematomas may be treated by internal drainage into the cisterna magna.

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SURGICAL TREATMENT OF SACCULAR INTRACRANIAL ANEURYSMS

A REPORT OF 56 CONSECUTIVELY TREATED PATIENTS

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IMPROVEMENT in technical aids available to the neurosurgeon has lowered the operative mortality for surgical treatment of intracranial vascular lesions. However, the operative mortality for intracranial aneurysms reported in the literature has continued to be high, especially for those treated by the intracranial approach. For this reason many surgeons have been content to ligate the carotid artery in the neck rather than assume an operative mortality and morbidity as great or greater than if the aneurysm were not treated at all. The purpose of this paper is to report a large series of surgically treated aneurysms of the circle of Willis. The majority of these lesions have been treated by direct intracranial attack. Only berry or saccular aneurysms are reported. The surgical results of treatment of arteriovenous anomalies and carotid-cavernous sinus fistulae will be reported in a separate article.

TABLE 1

Author	Total No. Cases	Op. Mortality %	No. Neck Ligations	Op. Mortality %	No. Intracranial Ops.	Op. Mortality %
Dandy[2]	36	25	1	100	35	26
Bassett et al.[1]	73	27	29	3.4	43	43
Poppen[6]	143	6.2	101	3	40	15
Hamby[4]	32	50	11	36	24	63
Falconer[3]	50	18	15	27	35	11
Murphey[5]	42	11.8	42	11.8	0	0
Hayes, Steelman, Rizzoli	56	3.6	14	0	42	4.7

REVIEW OF THE LITERATURE

Only authors who reported more than 30 patients surgically treated are cited. Their results are listed in tabular form in Table 1 together with those of the present authors for comparison.

Dandy's[2] original report of 36 patients surgically treated listed an opera-

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tive mortality of 25 per cent. One of his patients had only a ligation of the carotid artery in the neck, with resultant death. In 14 patients the aneurysm was trapped between intracranial clips and the carotid was ligated in the neck; there was 1 death (7 per cent) and 1 hemiplegic (7 per cent). In 21 cases various intracranial procedures were performed; there were 7 deaths (33 per cent) and 3 hemiplegics (19 per cent).

Bassett *et al.*[1] treated 73 patients by surgery with 20 deaths (27 per cent). Of 29 patients with cervical carotid ligations, 1 died (3.4 per cent) and 12 (41 per cent) were rendered hemiplegic. The trapping procedure was used on 18 patients; there were 9 deaths (50 per cent) and 2 hemiplegics (22 per cent). Twenty-five patients had an intracranial clipping of the aneurysm; there were 10 deaths (40 per cent) and 10 hemiplegics (66 per cent). Their operative mortality, therefore, for 43 patients having an intracranial operation was 43 per cent.

Hamby[4] listed an overall mortality of 50 per cent of 32 patients surgically treated. There were 11 cervical carotid ligations with a mortality of 36 per cent. Of 24 patients with intracranial operations 63 per cent died. There were 9 trapping operations, with a mortality of 22 per cent.

Falconer[3] treated 50 patients with aneurysm by surgery with an operative mortality of 18 per cent. Of 15 patients who had cervical carotid ligation only, 27 per cent died and of the 35 treated with an intracranial operation, 11 per cent died.

Poppen[6] has the largest series of aneurysms reported in the literature, and over two-thirds of his patients were treated simply by carotid ligation in the neck. In 101 cervical ligations there were 3 deaths (3 per cent) and 8 hemiplegics (8 per cent). It is of interest that 8 (8 per cent) of these patients have since died, 6 of them because of further rupture of the aneurysm. Of 18 patients subjected to trapping procedure 1 died (6 per cent). Of the 22 others who had intracranial procedures 5 died (23 per cent). Therefore the operative mortality in 40 cases of intracranial operation was 15 per cent.

Murphey[5] has treated aneurysms by cervical carotid ligation. He has ligated the carotid artery in the neck for aneurysms of different locations in 42 patients with a mortality of 11.8 per cent and 19 per cent hemiplegic morbidity.

MATERIAL

This series of operations for aneurysms was originally begun by one of us (H.V.R.) at Walter Reed Army Hospital in 1946 and continued there by the other authors (G.J.H. and H.F.S.). The operations at the other hospitals (George Washington University Hospital, Mt. Alto Veterans Hospital and Emergency Hospital, all at Washington, D. C.) were done by H.V.R. Although these aneurysms were treated at different hospitals by three different surgeons, the approach to the problem has been similar enough to make the series homogeneous. All patients treated surgically during the years 1946-52 are included.

The 2 postoperative deaths occurred within 72 hours of surgery. None

of the patients has since died nor had recurrence of symptoms. Hemiplegia that was present after operation and not before is listed as "postoperative hemiplegia." If hemiplegia was caused by the initial hemorrhage and not related to surgery, it was not counted as a postoperative morbidity. All aneurysms were verified by surgical exposure and/or arteriography.

METHOD OF TREATMENT

All of the patients had signs and symptoms manifested by an acute or subacute cerebrovascular episode, either with subarachnoid or intracerebral hemorrhage or paralysis of a cranial nerve. The clinical manifestations of aneurysm are well documented elsewhere and will not be repeated here. Neither surgery nor arteriography was performed in the acute phase of the illness, i.e. within the first 48 hours following the apoplectic attack. When the vital signs became stabilized and the state of consciousness improved, carotid arteriography was performed by percutaneous puncture (85 per cent) or direct exposure (15 per cent) of the carotid artery in the neck. If an aneurysm was shown on the films the decision whether to operate was made. No patients with negative arteriograms were operated upon. The following factors were considered in deciding for or against surgical attack upon the aneurysm: (1) Location of the aneurysm, (2) age of the patient, (3) size of the neck of the aneurysm, if present, (4) the general and neurological condition of the patient, (5) ability of the patient to tolerate carotid compression for 30 minutes (Matas test), and (6) the likelihood of producing an intolerable deficit if it were necessary at operation to sacrifice the artery feeding the aneurysm. No rigid criteria were adopted for any one situation, but the operative procedure for each patient was made flexible enough to produce maximal results with minimal risk.

Aside from the flawless execution of the surgical procedure the most important consideration is the surgeon's judgment of the proper time to operate. Since the clinical course of hemorrhage from intracranial aneurysm is notoriously unpredictable, the surgeon may have difficulty in selecting the optimal time for surgery. The time for surgery is that time when the vital and neurological signs have leveled off. If the surgeon operates before this time he will be subjecting the patient to an increased risk. If he waits too long for this optimal state to be reached, the patient may die before surgery can be done. The surgeon, therefore, must develop an acumen for timing the operation and furthermore have the necessary courage to undertake bold procedures when dealing with this deadly disease. As his ability to carry out the proper procedure at the proper time improves, the surgeon will be rewarded with a permissible operative mortality and morbidity. Until the surgeon can offer the patient a better chance for improvement with surgery than without, the patient should not be subjected to surgery.

The danger to life and neurological function is a calculated risk which must be assumed when the decision to operate is made. Furthermore, the outcome of surgery often depends on factors that cannot be forecast nor

avoided. For example, the rupture of an aneurysm, when exposed at operation, may mean the difference between a catastrophe and a successful surgical procedure. Before the surgeon should be willing to attack an aneurysm he must not only have every detail planned in advance, but he must be ready for any situation that may present itself.

RESULTS OF VARIOUS SURGICAL PROCEDURES

A total of 56 patients were treated by various surgical procedures. There were 4 (7 per cent) resultant hemiplegias (2 dominant, and 2 nondominant hemisphere) and 2 (3.6 per cent) deaths (Table 2). Of the 42 patients who had an intracranial procedure 3 had postoperative hemiplegia (7 per cent) and 2 died (4.7 per cent). Neither anticoagulants nor induced hypotension were used.

TABLE 2

Results of surgical procedures

Procedure	No. of Cases	Postoperative Hemiplegics		Postoperative Deaths	
		No.	%	No.	%
Ligation of carotid artery in neck	14	1	7	0	0
Trapping between intracranial clips and ligature in neck	17	2	12	0	0
Trapping between intracranial clips	13	1	6	2	15
Occlusion of neck of aneurysmal sac:					
With removal of aneurysm	3	0	0	0	0
Without removal of aneurysm	7	0	0	0	0
Packing with muscle and/or gelfoam	2	0	0	0	0
Total patients treated surgically	56	4	7	2	3.6
Total treated by intracranial operation	42	3	7	2	4.7
Total treated by neck ligation alone	14	1	7	0	0

Ligation of the Carotid Artery in the Neck. Of 14 patients none died and 1 became hemiplegic (dominant hemisphere). There were 7 ligations of the common carotid alone, 4 of the internal carotid and 3 of the common carotid followed by obliteration of the internal carotid. The only morbidity followed a one-stage ligation of the internal carotid. It is the opinion of the authors that ligation of the common carotid artery is safer than ligation of the internal carotid and that it is safer to ligate the former and follow with ligation of the latter in stages.

Trapping Between Intracranial Clip and Ligature on the Carotid Artery in the Neck. In 17 cases there were 2 hemiplegias (1 dominant, and 1 nondominant hemisphere) and no deaths. In 9 of the 17 cases the intracranial procedure was preceded by carotid ligation in the neck, and in 8 cases the operation was done in one stage.

Trapping Between Intracranial Clips. In this procedure the aneurysm is isolated from the circulation and it is, of course, necessary to sacrifice the

feeding artery. Thirteen patients were treated by this method with 1 resultant hemiplegic (6 per cent) and 2 deaths (15 per cent).

Occlusion of the Neck of the Aneurysmal Sac. Ten patients had aneurysms with a neck small enough and long enough to tolerate a metal clip. There were no deaths nor morbidity. In 3 of these the aneurysmal sac was removed and in 7 the sac was left in situ.

Packing or Reinforcing with Muscle or Gelfoam. There were only 2 instances of packing in this series and there were no deaths nor morbidity. This procedure is one that can seldom be planned deliberately before operation because the situations encountered during exposure may make it necessary to clip the aneurysm or its feeding artery. It is primarily of value in intact aneurysms that cannot be attacked radically because of their critica.

TABLE 3

Results of aneurysms in different locations

Location	No.	Procedure	No.	Mortality		Hemiplegia	
				No.	%	No.	%
Internal carotid artery, subclinoid	21	Carotid ligation in neck	8	0	0	0	0
		Trapping between intracranial clips and neck ligature	13	0	0	1	8
			21	0	0	1	4
Internal carotid artery, supraclinoid	20	Carotid ligation in neck	4	0	0	1	25
		Trapping between intracranial clip and ligature in neck	4	0	0	0	0
		Occlusion of neck of sac	5	0	0	0	0
		Trapping between intracranial clips	7	1	14	1	14
			20	1	5	2	10
Anterior cerebral artery	6	Occlusion of neck of sac	1	0	0	0	0
		Trapping between intracranial clips	5	1	20	0	0
			6	1	17	0	0
Middle cerebral artery	7	Carotid ligation in neck	2	0	0	0	0
		Occlusion of neck of sac	4	0	0	1	25
		Packing with muscle	1	0	0	0	0
			7	0	0	1	14
Posterior communicating artery	0						
Posterior cerebral artery	0						
Vertebral artery	1	Packing with muscle	1	0	0	0	0
Posterior inferior cerebellar artery	1	Trapping between intracranial clips	1	0	0	0	0

location. If the packing produces enough scar tissue, further rupture will be forestalled. If, however, the aneurysm bursts again the effects will be disastrous. There is no way of predicting whether the packing will prevent such a catastrophe.

RESULTS OF ANEURYSMS IN DIFFERENT LOCATIONS

The results and methods of specific treatment of aneurysms in different locations are listed in Table 3.

Internal Carotid, Subclinoid. Of a total of 21 patients, ligation of the carotid artery in the neck was sufficient to cause symptomatic and neurological improvement and prevent further bleeding in 8 patients with no mortality nor morbidity. In 13 patients the aneurysm was trapped between intracranial clips and a ligature on the carotid artery in the neck; there were no deaths and 1 hemiplegic (8 per cent). In 8 patients the intracranial operation was preceded by carotid artery ligation in the neck and in 5 patients the two ligations were done during one operation. In summary, there were 21 patients treated with no deaths and 1 hemiplegic (4 per cent).

Aneurysms in this location are best treated by ligation of the common carotid artery followed by ligation of the internal carotid artery 1 week later. These procedures are performed after compression of the vessel has been tolerated by the patient 30 minutes without undesirable sequelae. If the patient's age and suitability for craniotomy do not contraindicate, liga-

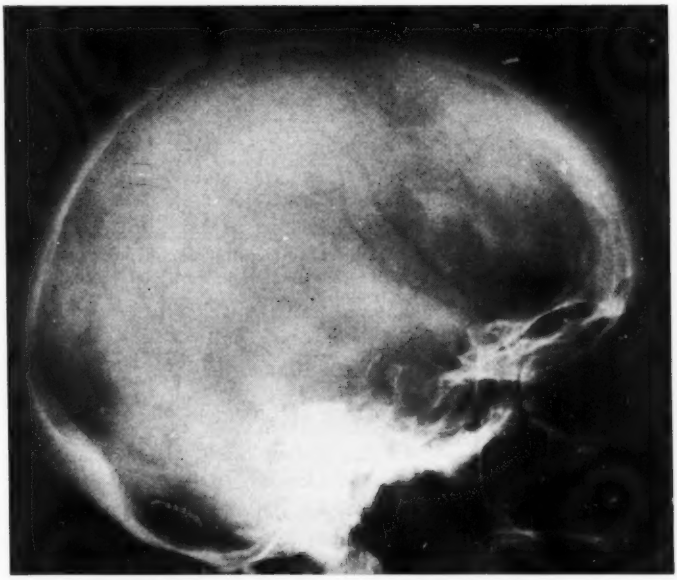

FIG. 1. *Case 1.* Plain x-ray of skull showing circular calcification in wall of subclinoid aneurysm of internal carotid artery. See Fig 2 for arteriogram.

tion of the carotid artery in the neck should be followed by clipping the internal carotid intracranially, in order to trap the aneurysm.

Case 1. A 72-year-old woman was seen in consultation because of pain over the left eye for 1 year. Three weeks before she was first seen she suddenly had an increase of the pain and the left eyelid drooped.

Neurological findings were hypalgesia over the 1st division of the left trigeminal nerve and a complete left 3rd nerve paralysis. X-rays of the skull showed a circular shell of calcification, 2.5 cm. in diameter, in the left parasellar region (Fig. 1). A left

FIG. 2. *Case 1.* Internal carotid arteriogram (diodrast) showing large aneurysm of internal carotid artery. Regression of symptoms and signs followed ligation of internal carotid artery in the neck.

carotid arteriogram (May, 2 1949) showed that this calcification was the wall of an aneurysm on the subclinoid portion of the internal carotid artery (Fig. 2).

The left internal carotid artery was occluded for 30 minutes under local analgesia 5 days later and then ligated and divided.

There was rapid improvement of her ophthalmoplegia and headache. Four years later she was alive and well.

Internal Carotid, Supraclinoid (20 Patients). In 4 patients carotid ligation in the neck only was necessary with no deaths and 1 hemiplegic. Trapping of 4 aneurysms between intracranial clips and carotid artery ligature in the neck was done without mortality or morbidity; of these 2 were done at one operation. The other procedures used were occlusion of the neck of the sac (5 patients) and trapping between intracranial clips (7 patients). The latter

procedure caused 1 death (14 per cent) and 1 hemiplegic (14 per cent). In summary, of 20 patients treated by various surgical procedures 1 died (5 per cent) and 2 were hemiplegic (10 per cent).

The treatment of supraclinoid aneurysms is similar to that of infraclinoid ones with the exception that they can be attacked intracranially more easily since they are located farther up on the carotid tree. Again, initial treatment is ligation of the carotid in the neck unless the neck of the aneurysm and its location render it suitable for clipping without sacrificing the carotid artery.

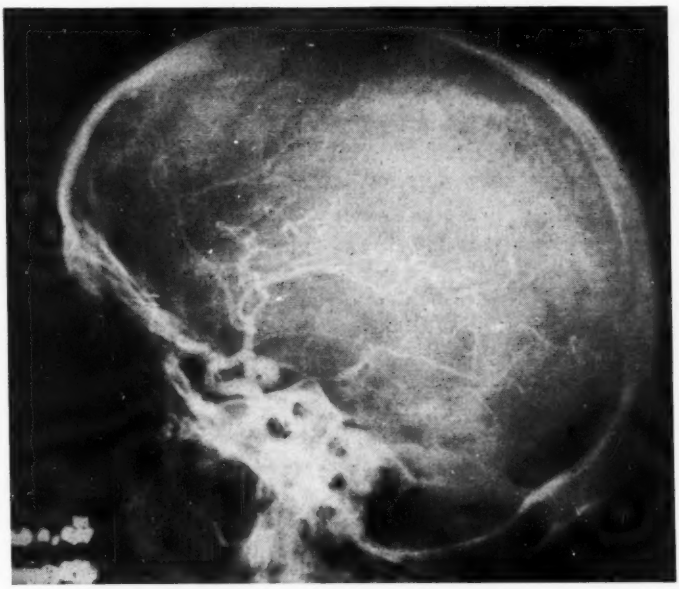

FIG. 3. *Case 2.* Internal carotid arteriogram (diodrast). There is an aneurysm arising from supraclinoid portion of internal carotid artery. Upward displacement of middle cerebral artery indicates presence of intratemporal lobe hematoma. Craniotomy was done and the aneurysm was trapped between clips placed on the carotid artery. Subdural and intracerebral hematomas were evacuated. Satisfactory result.

In this instance, if the intracranial approach is done first, the carotid artery should be exposed in the neck before the intracranial approach is done. By thus being prepared for unexpected bleeding, the surgeon can then trap the aneurysm if clipping of its neck is not successful. If preliminary ligation of the carotid artery in the neck produces no adverse effects and the surgeon feels that an intracranial procedure can be undertaken with minimal risk, the aneurysm should be exposed intracranially and the carotid artery clipped distally to the aneurysm, thus trapping it and effecting a cure. If, however, the aneurysm lies too closely to the bifurcation of the carotid to admit the entrance of a clip, the surgeon should be content with cervical carotid liga-

tion rather than risk the intracranial approach which may require clipping of the anterior cerebral and possibly the posterior cerebral artery.

Case 2. A 23-year-old girl had been well until July 18, 1950, when there was a sudden onset of severe right frontal headache and drooping of the right eyelid. Examination showed bilateral early papilledema, a complete right 3rd nerve paralysis and a left central facial weakness.

She was admitted to the George Washington University Hospital on July 20, 1950. On July 22, a right carotid arteriogram showed a 1 cm. saccular aneurysm arising from the supraclinoid portion of the internal carotid artery (Fig. 3). There was displacement of the middle cerebral artery upward, indicating the presence of an intratemporal lobe hematoma.

On Aug. 9, 1950, a right frontotemporal craniotomy was performed. There was a subdural hematoma, 1 cm. thick, over the anterior portion of the temporal lobe. An intracerebral hematoma was evacuated from the temporal lobe and immediate access to the aneurysm was obtained. The aneurysm was trapped by placing clips on the carotid artery proximal and distal to the aneurysm.

Her postoperative course was uneventful. Her subjective complaints and objective neurological signs disappeared. She was later shown to have polycystic kidneys. Except for hypertension she was well $2\frac{3}{4}$ years after surgery.

Anterior Cerebral Artery (6 Patients). In 1 patient the neck of the aneurysm was occluded (dominant hemisphere) without mortality or morbidity. In 5 patients the aneurysm was trapped between intracranial clips, with 1 (20 per cent) death (dominant hemisphere). In the other 3 patients the aneurysm was in the nondominant hemisphere and there was no postoperative morbidity. In summary, 6 aneurysms were treated with 1 death (17 per cent) and no morbidity.

Aneurysms on the anterior cerebral artery are probably not affected by ligation of the carotid artery, and none of the patients had this procedure done. The operability of these aneurysms is determined by the size of the neck and its relationship to the anterior communicating artery. If the lesion is located on the anterior communicating artery between the two anterior cerebrals and fills from both sides by arteriography, it probably should not be operated on, since the surgeon may be forced to sacrifice both anterior cerebrals with either postoperative death or with a pitiable mental and neurological deficit. The risk of surgery here is as high as or higher than the risk of leaving the aneurysm alone. The surgeon is well-advised to leave these alone.

Aneurysms of the anterior cerebral artery seldom have a small enough neck to tolerate a clip and the surgeon must usually sacrifice the feeding artery. It was necessary to do this in 5 out of the 6 cases in this group. There is no way of predicting in advance whether the patient will tolerate sacrificing one anterior cerebral artery. The risk to life and neurological function of sacrificing the left (dominant) anterior cerebral artery is greater than that on the right, and this must be given considerable weight when the surgeon is contemplating attacking an aneurysm in this location.

Case 3. On Mar. 24, 1951, a 21-year-old soldier suddenly had headache and dizziness and lapsed into coma while on duty in Italy. A lumbar puncture yielded grossly bloody fluid under elevated pressure. His condition gradually improved and he was evacuated to the United States.

Following admission to Walter Reed Army Hospital no localizing neurological signs were present. A bilateral carotid arteriogram showed a large (1 cm.) aneurysm on the right anterior cerebral artery (Fig. 4).

FIG. 4. *Case 3.* Internal carotid arteriogram (AP projection, diodrast). Saccular aneurysm on the right anterior cerebral artery was trapped between clips. Satisfactory results.

On June 26, 1951, a right frontal craniotomy was performed but the aneurysm was not exposed because of cerebral edema.

On July 6, 1951, the bone flap was reelevated and the aneurysm was found arising from the anterior cerebral artery proximal to the communicating artery. It was trapped between clips and coagulated.

The postoperative course was uneventful and he was returned to duty 3 months later without complaints or disability.

Middle Cerebral Artery (7 Patients). Two patients had a ligation of the carotid artery in the neck and have since had no further difficulty for a period of 6 weeks and 3 years respectively. In 1 patient the aneurysmal sac was packed with muscle after an intracerebral hematoma had been evacuated from the temporal lobe. In the remaining 4 patients the neck of the aneurysm was occluded leaving the main trunk intact. In 3 of these patients there was no sequela. In 1 patient a hemiplegia and aphasia developed immediately postoperatively; at that same operation an intracerebral hematoma had been evacuated. He eventually made an excellent recovery from

both hemiplegia and aphasia and it is unlikely that he actually had a complete thrombosis or ischemia of these areas irrigated by the middle cerebral artery.

The decision for treatment of a middle cerebral artery aneurysm requires excellent arteriograms in both anteroposterior and lateral projections to determine its exact position and size of the neck. If the aneurysm is located on the left (dominant) middle cerebral artery or if the neck is too broad to take a clip, then ligation of the carotid artery in the neck is all that should be done. If the neck looks as if it will take a clip then operative exposure and clipping are justified when it is located on the nondominant hemisphere. This procedure requires a perfection of operative technique and unless the operation can be performed under ideal conditions, then simple ligation of the carotid is the wise approach.

If an intracerebral hematoma is present, as evidenced by displacement of blood vessels in the arteriogram, then an osteoplastic craniotomy should be done to evacuate the hematoma. A bone flap opening is better than a burr hole because the surgeon will probably need the added room if he be forced to clip the source of the bleeding once the clot is sucked out.

Case 4. A 24-year-old soldier had a sudden onset of frontal headaches on May 3, 1952. These headaches improved until 6 weeks later when they recurred and he lapsed into coma for 2 days. A lumbar puncture showed grossly bloody fluid under a

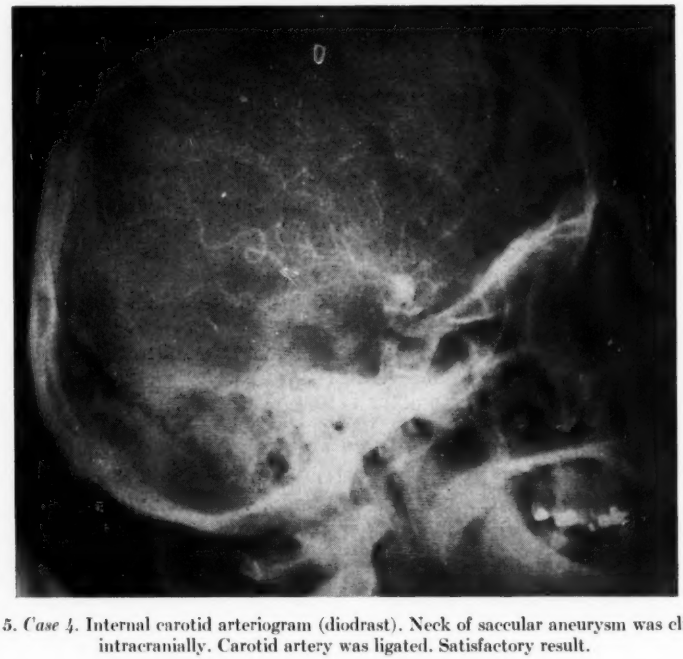

FIG. 5. *Case 4.* Internal carotid arteriogram (diodrast). Neck of saccular aneurysm was clipped intracranially. Carotid artery was ligated. Satisfactory result.

pressure of 360 mm. of water. He was evacuated to the United States from Japan, arriving at Walter Reed Army Hospital Aug. 3, 1952.

On examination the positive signs were drowsiness, bilateral papilledema and slight left facial weakness of the central type. A right percutaneous carotid arteriogram on August 8 showed a berry aneurysm, 1 cm. in diameter, on the proximal part of the middle cerebral artery (Fig. 5). Matas test was negative.

On Aug. 13, 1952, the aneurysm was exposed and the neck was clipped, sparing the middle cerebral trunk. The carotid artery was also clipped intracranially.

Postoperatively he did well. The only neurological deficit present 6 months after operation was a left central facial weakness. He was discharged from the Army at the termination of his enlistment.

Posterior Communicating and Posterior Cerebral Arteries. None of the aneurysms was located on these vessels.

Vertebral Artery. The 1 case of aneurysm of the vertebral artery in our series is presented below:

Case 5. A 30-year-old woman complained of intolerable bruit in her head and left occipital headaches of 3 years' duration. These symptoms had become progressively worse.

Neurological findings were negative, except that a bruit synchronous with the pulse beat was heard over the left suboccipital region.

On Mar. 19. 1947, a unilateral exposure of the left cerebellopontine angle was made and a large aneurysm of the vertebral artery was found. It was reinforced with muscle and no further treatment was done.

Postoperatively she was relieved of her complaints (probably because of the bony decompression) and has continued well during the 5 years since operation.

Posterior Inferior Cerebellar Artery. There was 1 aneurysm located on this vessel and the history is as follows.

Case 6. A 26-year-old soldier was admitted to Walter Reed Army Hospital Dec. 16, 1946, with a history of having been unconscious after a fight the night previously.

On admission he was semicomatose. He had bilateral internal strabismus, bilateral retinal hemorrhages, left-sided Babinski toe signs and stiff neck. A lumbar puncture yielded bloody spinal fluid under a pressure of 400 mm. water.

During the next 3 weeks he became more alert but continued to be drowsy, and saw double. Marked bilateral ataxia of the extremities and papilledema developed. The spinal fluid continued to be bloody and the pressure high.

On Jan. 16, 1947 bilateral exploratory trephinations were made in the occipital and temporal regions with negative findings. On January 20 a ventriculogram showed internal hydrocephalus with shift of the 4th ventricle to the right.

On Jan. 30, 1947 a suboccipital craniotomy was done and an aneurysm, 2 cm. in diameter, was found arising from the posterior inferior cerebellar artery.

FIG. 6. *Case 6.* Photograph of aneurysm removed from posterior inferior cerebellar artery. Six-year followup with satisfactory result.

The lesion arose from the artery distal to the medullary branches and, therefore, the neck was clipped and the aneurysm was removed (Fig. 6).

Recovery was slow and he required prolonged physical rehabilitation. He was discharged from the Army because of physical disability. A followup 6 years later revealed he was working full time as a mechanic and had no subjective complaints.

SUMMARY

1. Fifty-six consecutive patients with surgically treated intracranial aneurysms are reported. The overall surgical mortality was 3.6 per cent and 7 per cent of the patients had a postoperative hemiplegia.

2. Of 14 patients treated by ligation of the carotid artery in the neck, none died and 1 patient (7 per cent) was rendered hemiplegic.

3. Of the 42 patients requiring an intracranial operation, 2 died (4.7 per cent) and 3 became hemiplegic (7 per cent).

4. Factors that make for success or failure in the surgery of aneurysms are listed and discussed.

5. A rational method of treatment of aneurysms in different locations based on successful treatment is outlined.

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STUDIES OF PRESSURES IN THE CAROTID ARTERY OF PATIENTS UNDERGOING CEREBRAL ANGIOGRAPHY*

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THE success of surgical treatment of intracranial arterial lesions, such as aneurysms, arteriovenous malformations, shunts, and vascular tumors, depends in part upon the ability to occlude major arterial channels in the neck and intracranial space. Such occlusion can be followed by transient and permanent neurologic deficit. Studies designed to prognosticate the occurrence and severity of such complications are in demand.

Sweet and Bennett[4] in 1948 presented the first series of internal carotid artery pressure determinations before and after ipsilateral and simultaneous bilateral carotid artery occlusion. They suggested the usefulness of data on pressures during bilateral occlusion in determining the patency of the main cross-over channels of the circle of Willis. They further suggested the use of angiography together with recordings in assessing the effectiveness of the distributing mechanism of the circle of vessels at the base of the brain.

By studying intracarotid arterial pressures and relating them to the radiologic patterns of arterial filling, we have attempted to obtain further data as to the adequacy of the distribution mechanism, whether it be anatomic, metabolic, neurogenic, or, as is probably nearer the truth, a combination of these. We hoped to be able to prognosticate from the angiographic studies how much residual pressure would be expected after carotid artery occlusion.

METHODS OF STUDY

In 10 patients who were under study for various neurologic disorders, intracarotid arterial pressures were recorded with the aid of a Sanborn strain gage amplifying and recording instrument from a Statham transducer with a range of 0–75 cm. of mercury. A No. 18 needle was placed into the exposed common carotid artery, and a continuous blood pressure record was obtained before and during ipsilateral and bilateral common carotid arterial occlusion at a point proximal to the puncture site. (Pressures during occlusion are expressed as per cent of original pressures.) Thereafter bilateral

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common carotid arteriography was carried out by the use of 35 per cent Diodrast. Both anteroposterior and lateral projections were made on each side with the aid of a serial cassette changer.

Four additional patients were studied by a more limited technique in which either the observations during occlusion or the arteriographic visualizations were obtained unilaterally.

RESULTS

The data on the 14 patients are presented in Table 1.

TABLE 1

*Residual arterial pressure percentages after unilateral and bilateral carotid artery compression compared with evidences of angiographic filling of the two anterior cerebral arteries and the posterior cerebral artery ipsilaterally**

Patient	Diagnosis	Side Measured	Residual Pressure % From		Arteriographic Findings			
					Right Injection		Left Injection	
			Ipsilateral Compression	Bilateral Compression	Ant. Fill. Bilateral	Posterior Filling	Ant. Fill. Bilateral	Posterior Filling
H.B.	Not established	L	62/76	15/24	+	+	+	0
H.B.	Not established	R	72/88	64/82	+	+	+	0
M.B.	Idiopathic epilepsy	L	70/89	26/40	+	0	+	0
L.K.	Not established	L	61/78	50/72	+	+	0	+
H.R.	Not established	R	61/76	43/50	not injected		+	0
R.C.	Subarachnoid hemorrhage	R	50/67	50/67	+	0	0	0
R.H.	Frontal tumor	R	52/70	31/45	not injected		0	+
A.L.	Birth injury epilepsy	R	50/67	33/44	0	0	0	0
A.L.	Birth injury epilepsy	L	46/63	31/42	0	0	0	0
G.T.	Cerebral thrombosis	L	50/62	27/33	0	0	0	0
R.A.	Multiple aneurysms	R	33/64	20/36	0	+	+	+
J.C.	Carotid thrombosis	R	40/53	no data	0	0	not injected	
J.C.	Carotid thrombosis	L	35/55	no data	0	0	not injected	
J.G.	Subarachnoid hemorrhage	L	38/63	19/31	+	0	+	+
J.M.	Carotid thrombosis	R	33/50	no data	+	0	0	0
W.B.	Carotid-caver. fistula	R	26/40	16/25	0	0	+	0
G.S.	Subarachnoid hemorrhage	R	21/31	8/12	+	0	+	0

* The residual pressure percentages are arranged in order of decreasing values. Note the lack of corresponding change in the evidence of angiographic filling.

A. *Ipsilateral Common Carotid Artery Occlusion: 14 cases.* Unilateral occlusion produced a residual pressure range extending from 21/31 per cent to 70/89 per cent or, expressed in terms of mean arterial pressures, from 25 to 80 per cent.

In 6 cases both systolic and diastolic residual pressures were 50 per cent or more of the original values, and in 6 cases the residual systolic pressure was less than 50 per cent but the diastolic pressure was 50 per cent or more of the control measurements. Thus in 12 of the 14 cases the diastolic pressure remained at 50 per cent or more of the control pressure.

At the time of angiography 8 of these 12 patients demonstrated bilateral filling of the anterior half of the cerebral circulation from either one or both sides of the circle of Willis.

Two patients in whom injections were made on both sides showed no bilateral filling from either side, and the last 2 patients who received injections only on one side failed to show bilateral filling from the single side studied.

In 2 cases the residual diastolic and systolic pressures were less than 50 per cent of the control pressures. Both of these patients showed bilateral filling of the anterior half of the circulation.

In each case in which the residual systolic pressure was 60 per cent or more and the residual diastolic pressure 75 per cent or more (4 cases), bilateral filling of the anterior half of the circle from one or both sides was observed. Below these levels of residual pressure there was found to be no correlation with the filling of the circulation as seen by angiography (Fig. 1).

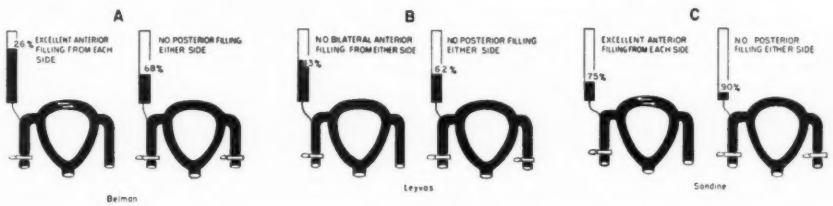

FIG. 1. Comparison of the percentage of pressure fall after unilateral occlusion with the evidence of arteriographic filling. (A) Mild fall and excellent bilateral filling. (B) Intermediate fall and no bilateral filling. (C) Severe fall and excellent bilateral filling.

B. *Bilateral Common Carotid Artery Occlusion: 12 cases (14 observations).* Six observations were made on 4 patients. Pressures were recorded on each side as a separate series of observations in 2 patients and in 2 patients only unilateral angiography was performed. After simultaneous bilateral occlusion the residual pressure percentages ranged from 8/12 per cent to 50/72 per cent. The range of mean arterial pressures was 10 to 61 per cent.

All patients were divided into 3 groups according to their pressure readings. The results of angiographic studies to visualize the posterior half of the cerebral circulation were recorded for each group.

Group 1 was made up of 3 patients who showed residual systolic and diastolic pressures of 34 per cent or more. Two of these demonstrated posterior cerebral artery filling on one or both sides.

Group 2 included 5 patients with residual pressures, both systolic and diastolic, of less than 34 per cent of the control observations. Three patients in this group showed no posterior cerebral artery filling on either side.

Group 3 included 4 patients with residual systolic pressures of less than 34 per cent and diastolic pressures of 34 per cent or more. Of these patients with intermediate pressures, 2 demonstrated posterior filling on one or both sides and 2 showed no filling.

COMMENT

A static diagram of the circle of Willis gives little clue as to what residual pressures may be expected after the occlusion of one or both of the carotid systems that contribute blood to the brain. We observed marked variation from one patient to the next. The angiographic studies in this series gave little additional aid in estimating the probable residual pressures after occlusion of one or both carotid arteries. Of the blood reaching the circle of Willis the percentage that is contributed by the vertebrobasilar system is unknown in man. The studies of Dumke and Schmidt[2] showed that in monkeys this system contributed from 26 to 80 per cent of the amount of blood carried by the combined carotid systems. If both common carotid vessels are occluded, the residual pressure might be expected to reflect, in part at least, the adequacy of such a contribution through the basilar artery. In the data collected in this study there was no correlation between the residual pressures and angiographic demonstration of filling of the posterior half of the circulation (Fig. 2) nor could the presence of bilateral filling of

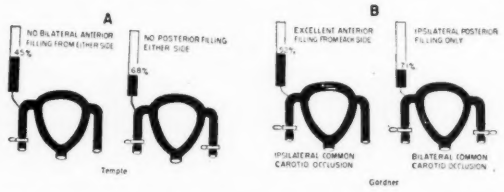

FIG. 2. Comparison of the percentage of pressure fall after bilateral occlusion. (A) Intermediate pressure fall and no posterior filling. (B) Similar degree of fall and ipsilateral posterior filling.

the anterior circulation be correlated with the residual pressures observed with the exception that after unilateral occlusion the preservation of high systolic residual pressures (60 per cent or more) and high diastolic pressures (75 per cent or more) was associated with evidence of bilateral filling of the anterior half of the circulation. However, given the series of angiographic studies with evidence of cross-over between the two halves of the circulation, no reliable prediction could be made of the residual pressure to be observed. Thus in 4 patients (5 observations) the anterior half of the circulation filled

bilaterally from each side injected, and the residual pressure percentages observed were at the two extremes of the table. On the other hand, neither did the absence of bilaterality of filling of the anterior half of the circulation or of filling of any part of the posterior half of the circle show any predictable relationship to the residual pressures. Brackett and Mount[1] in 1951 reasoned that if the residual internal carotid arterial pressure after ipsilateral carotid artery occlusion were not materially reduced further by contralateral occlusion, then it could be assumed that no significant anterior circulation existed. This opinion was given support in 1953 by Strobos and Mount,[3] who compared the data on residual pressures after unilateral and bilateral carotid artery occlusion with angiographic studies. By using contralateral carotid vessel occlusion during the injection of the ipsilateral vessel, they observed the presence or absence of bilateral angiographic filling. The absence of cross-over of opaque material was interpreted as signifying deficient collateral circulation. Large falls in pressure after ipsilateral carotid occlusion correlated well with such demonstrations of "deficient" cross-over circulation as well as with an increasing incidence of complication after carotid ligation.

The arterial tree is not a rigid structure of branching and connecting tubes, and one must resist utilizing the attractive but all too simple analogy between the circle of Willis and a mechanical anastomosis. The elasticity of the arterial walls, the responsiveness of these walls to chemical stimuli, the possible neurogenic influences, and the effects of systemic blood pressure are all disregarded in such an analogy. Furthermore, the method of angiography employed is rather crude. The volume of opaque material that is injected varies in its relation to the capacity of the circulatory bed being studied. The pressure with which it is injected is also a variable. These factors must influence the degree of filling of the components of the arterial tree. In view of the known fallibilities of arteriographic techniques in consistently filling the same vessels in repeated injections, we cannot accept single angiographic records as being wholly reliable in reflecting the functional state of arterial cross-over.

Although the methods of Strobos and Mount come closer to a practical assessment of adequacy of functional cross-over, these authors caution that the reliability of the method is not complete. Another word of caution needs emphasis in our interpretation of such studies. While we may gain valuable information relating to the adequacy of the cerebral circulation by arterial pressure measurements, arterial pressure is not synonymous with cerebral blood flow. It is probably the most important factor in the maintenance of blood flow but must not be taken as the sole index of the adequacy of such flow. The factors determining cerebral vascular resistance also influence blood flow, and these are not taken into account in the measurement of arterial pressure.

SUMMARY AND CONCLUSIONS

Studies have been made of the intracarotid arterial pressure during ipsilateral and bilateral carotid artery occlusion. The data have been analyzed

with respect to their correlation with angiographic demonstration of filling of the anterior and posterior portions of the circle of Willis and evidence of bilateral filling from unilateral injections. The number of observations is small, but with the methods used no reliable correlation has been observed between the amount of pressure fall after ipsilateral and bilateral carotid arterial occlusion and the presence or absence of bilateral filling of the anterior half of the circle of Willis or the filling of the posterior half ipsilaterally. The only exception to this is the association noted between the preservation of high residual systolic and diastolic pressures after unilateral carotid occlusion and the presence of bilateral filling of the anterior cerebral artery. The use of angiography alone gives insufficient information from which to predict the adequacy of the mechanism of distribution in the circle of Willis.

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OSTEITIS DEFORMANS WITH SPINAL CORD COMPRESSION

REPORT OF THREE CASES

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APPARENTLY, osteitis deformans occurred in antiquity. Paleontologic evidence exists which, it is claimed, reveals manifestation of the disease in fossil apes.[10] Some observers[2] have felt that preserved specimens of prehistoric man show the characteristic alterations. It remained, however, for Paget to describe the disease as an entity.[11]

Justifiable therapeutic pessimism usually attends the diagnosis of osteitis deformans. Specific therapy is not available and palliative treatment is unrewarding. However, a syndrome occasionally accompanies Paget's disease that offers some hope of symptomatic relief for variable periods of time. This entity is spinal cord compression secondary to vertebral osteitis deformans. Three patients with the disease, manifesting signs of progressive spinal cord involvement, have been under our care and surgical decompression was accomplished in each by laminectomy.

REPORT OF CASES

Case 1. A 66-year-old white male was admitted to the hospital complaining of progressive weakness of both legs with burning pain of 2 years' duration.

Examination disclosed a "steppage" gait with atrophy of the musculature of the left leg. There was no definite sensory level although the joint-muscle-tendon sense was lost to the iliac crest. Roentgenograms revealed the characteristic changes of osteitis deformans in the dorsal spine and right hip joint (Fig. 1).

Since Pantopaque® myelography delineated a complete block of the subarachnoid space at the 8th dorsal intervertebral disc, a decompressive laminectomy was performed. The laminae

FIG. 1. *Case 1.* Osseous proliferation along ventral circumference of vertebral bodies and loss of bony detail are evident in dorsal spine.

* Aided by the Kresge Fund.

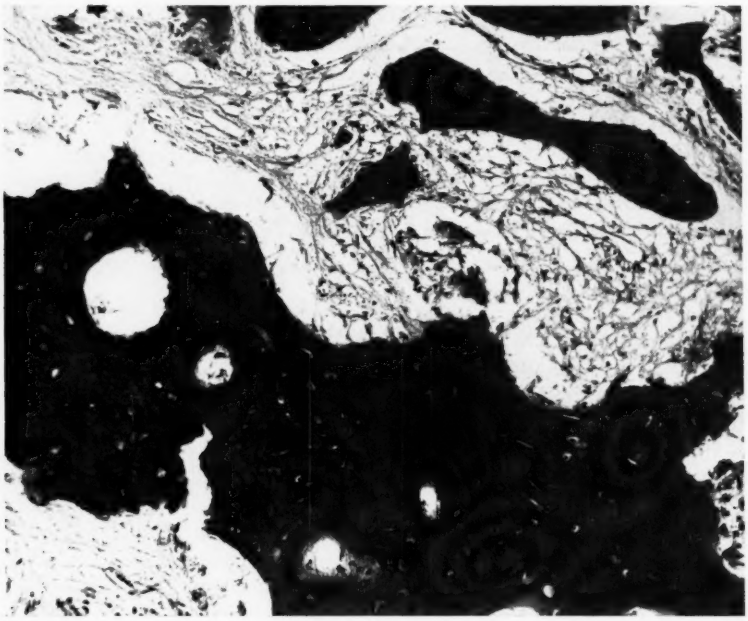

FIG. 2. *Case 1.* Photomicrograph of dorsal lamina showing typical trabecular mosaic bone with fibrosis of marrow. (Hematoxylin and eosin stain, ×90)

were extremely thick and vascular (Fig. 2). The patient remained asymptomatic for 1 year when the syndrome again appeared. Laminectomy was again performed at the same level and the pathology observed was similar. However, postoperatively, there was little improvement and the patient remains confined to a wheelchair 3 years after surgery.

Case 2. A white male, aged 65 years, had fallen from a stepladder 1 year prior to admission. Since that time he had noted numbness of both feet. Gradually, weakness of both legs appeared and on admission he was paraplegic with loss of all sensory modalities below the 7th dorsal dermatome.

After demonstration of an obstruction opposite the 5th dorsal vertebral body by myelography, decompression was undertaken. The laminae were 5 cm. in thickness and bled profusely.

Convalescence was remarkable in that within 6 months he was playing golf regularly. Five years later, however, pain and weakness of the legs reappeared. Radiographs revealed extensive osteitis deformans of the skull (Fig. 3) and the entire vertebral column. Myelographic block was relieved by decompression and the pathology was similar to that previously observed (Fig. 4). The patient is again walking unaided.

Case 3. A 34-year-old white male had sudden development of progressive weakness of both legs 6 months prior to hospital entry.

Examination disclosed a left "foot drop," weakness of all motions of both lower extremities, hypoactive patellar and Achilles reflexes, and a level of sensory impair-

FIG. 3. *Case 2.* Characteristic "cotton wool" appearance of skull.

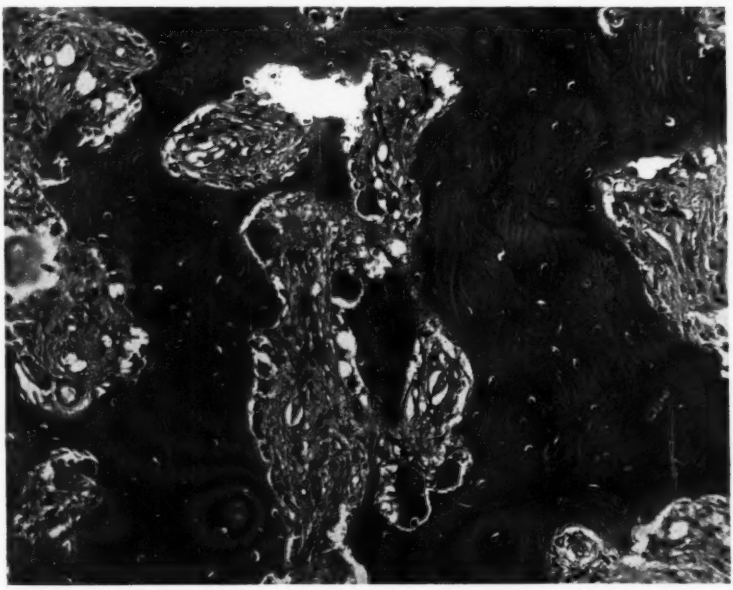

FIG. 4. *Case 2.* Photomicrograph of dorsal lamina showing heavy trabeculae with fibrotic marrow. (Hematoxylin and eosin stain, ×90)

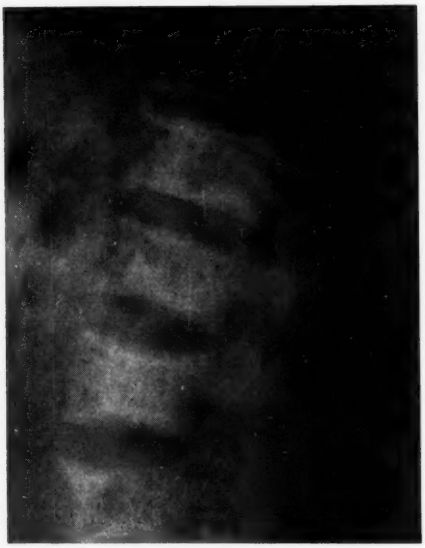

FIG. 5. *Case 3.* Areas of bony condensation and disintegration in lumbar spine.

ment from the 7th dorsal dermatome downward. Extensive osteitis deformans was revealed by skeletal survey x-rays (Fig. 5). Introduction of contrast medium via the cisterna magna outlined a complete obstruction at the 7th dorsal segment.

Surgical exposure of the area disclosed thick, vascular laminae which were removed. A diagnosis of osteitis deformans was made on the specimens of bone submitted.

Results of surgery were disappointing and the patient remains a custodial problem 3 years later, confined to a wheelchair by weakness of the extremities.

GENERAL CONSIDERATIONS

Osteitis deformans with spinal cord compression is a relatively rare syndrome. While Paget's disease has been found in 3 per cent of autopsies on persons over 40 years of age[13] there have been only about 50 cases of spinal cord compression secondary to the disease reported in the literature.

Since it has been hypothesized that mechanisms other than direct compression may produce spinal cord involvement in osteitis deformans, i.e. ischemia of the cord caused by either arteriosclerosis of spinal arteries[14] or foraminal narrowing with resultant circulatory embarrassment, it cannot be assumed that all patients with osteitis deformans and signs of spinal cord disease suffer from myelic compression. Therefore, perusal of literature[3,5–9,12,14,16–19] pertaining to the entity led us to establish certain criteria for the diagnosis and to exclude those cases that did not fulfill these requirements. We considered it absolute evidence that there was encroachment on the spinal cord if a myelographic block was demonstrated or if the pathologic process was verified by surgery or autopsy. It was regarded presumptive evidence if there was a subarachnoid block as demonstrated by lumbar puncture with jugular compression or if the protein of the cerebrospinal fluid was elevated. It is probable that in some of the cases not included there was, in fact, encroachment but the proof was not indisputable. We wish to consider, then, only 22 of the cases included in these papers.

These cases, on the whole, fall into a clinical pattern. Males predominated and the highest incidence was in the 4th through the 6th decades. Anamnesis usually revealed the steadily progressive course of spinal cord involvement, 1 to 2 years in duration. As stated by Turner,[17] the patient's

complaints are those of neurological impairment and not of the bone disease. In the great majority of the cases the initial complaint was impairment of sensation. Usually paresthesias appeared in the lower extremities; numbness, burning or "cramping" were included. Pain was also a frequent complaint. When present, the discomfort began in the vertebral column later to radiate into the extremities. In 11 of the 22 cases "girdle" sensations appeared but were not of great value in predicting the exact level of the lesion. In some cases, however, the syndrome began with progressive weakness in the involved limbs. As the disease developed, the gait became affected, varying from "shuffling" to definite ataxia. In 3 of the patients once the weakness had appeared the paralysis became rapidly complete. Trauma was not a significant precipitating factor.

Examination revealed that the usual stigmata of Paget's disease were not a necessary accompaniment. Since in all but 2 of the cases the level of involvement was in the upper dorsal area, hyperactive deep reflexes in the lower extremities with varying degrees of weakness in the legs were the rule. A sensory level corresponding roughly to the vertebral involvement was usually in evidence but the line of demarcation was by no means a sharp one.

Survey of the laboratory studies performed disclosed, as would be anticipated, that the protein content of the spinal fluid was elevated and that while the serum calcium, phosphorus and acid phosphatase remained normal, the alkaline phosphatase level was raised.

From a radiographic standpoint, spinal cord compression did not occur in the monostotic form of osteitis deformans in the cases under consideration. The patients who had received adequate roentgenologic investigation presented alterations in two or more adjacent vertebral segments with or without compression fractures, and demonstrable lesions in other portions of the skeleton. The great majority were classed as advanced involvement.

The operative findings were essentially the same in all cases. When exposed the laminae were extremely thick although easily removed with a rongeur. The bone was very vascular and bled profusely. After complete decompression by laminar excision, the dura mater appeared normal and was not opened in most of the cases. It was usual to observe the dura mater begin to pulsate freely after the constricting bony ring was removed. In all operated patients, save 1,[8] the compression was found in the mid-dorsal area. The explanation for this predilection may lie in the fact that since the interpedicular space is narrowest in the thoracic region it follows that the cross-sectional area of the spinal canal is at its smallest. Therefore, any proliferation of the bony vertebral ring would be more apt to manifest itself by spinal cord embarrassment at this level. This hypothesis has been advanced by Schwarz and Reback.[14]

Of the 22 patients, 16 were operated upon. There were 3 deaths in this group of surgical cases; 12 patients were considered benefited by the procedure. The amount of improvement varied from complete, but temporary,

recovery up to periods of 11 years to partial relief of symptoms for shorter periods of time.

DISCUSSION

In the light of the above presentation our cases conform to the general pattern. All were males and 2 were over 60 years of age. Case 3 is the exception. At 34 years of age he probably represents one of the youngest individuals in the literature of the syndrome.

The symptomatology of the 3 patients was the manifestation of progressive spinal cord involvement, and trauma appeared to be a precipitating factor in 1 case. Upon examination, they revealed varying degrees of incapacity. Case 1 was ambulatory with a "steppage" gait while Case 2 was bedridden with complete paraplegia. The occurrence of this type of gait in Case 1 and hypoactive tendon reflexes in the lower extremities of Case 3 appears paradoxical in the presence of a cord lesion in the thoracic region. However, it is possible that they were the product of a second lesion. Perhaps foraminal overgrowth in the lumbar area produced compression of peripheral nerves at this level with resultant hypoactive deep reflexes and weakness of dorsiflexion of the feet. The clinical picture may then have represented a composite of both pathological processes.

Laboratory studies in these cases merit little additional comment. Suffice it to say that they fell within the limits anticipated in osteitis deformans.

Roentgenograms in our patients demonstrated that involvement in all was of the polyostotic variety. Myelography delineated a complete block of the subarachnoid space in each. The level of obstruction was mid-dorsal in 3 cases. In all respects the radiographic findings paralleled closely those observed by other authors.

The appearance of the thickened laminae and the soft, vascular structure of the bone removed in conjunction with its microscopic appearance leaves little room for doubt that this was osteitis deformans. The difficulties in achieving adequate hemostasis in the 3 patients confirms the observation made by Edholm, Howarth and McMichael[4] who measured the blood flow through bone affected by osteitis deformans and found it increased many times that in normal bone.

The results of surgery were gratifying in 2 of the 3 cases. Case 1 was afforded symptomatic relief by the first procedure for 1 year. Case 2, initially the most severely affected, was able to resume nearly normal activity for a period of 6 years and is again improving after a second decompression. Case 3, however, was benefited only a short time by the operation and is now incapacitated.

CONCLUSIONS

From the information gained from the cases reported by other authors and the personal experience afforded by 3 cases it is our belief that a definite program of management should be adhered to in spinal cord compression secondary to osteitis deformans. All patients with a history suggesting pro-

gressive spinal cord involvement in whom Paget's disease is suspected should be investigated by myelography. Inability to perform lumbar puncture should not be a deterrent since the medium may be injected via the cisternal route. If an obstruction is encountered in the subarachnoid space the area should be adequately decompressed by laminectomy. We do not stand with Adson[1] who believed that "it is doubtful whether decompression of the spinal cord is indicated, since the relief obtained is but temporary"; nor do we believe that the severity of initial neurological deficit is a reliable guide as to the ultimate benefit from the surgery.

SUMMARY

Three cases of osteitis deformans with spinal cord compression are presented; 1 patient was aged 34 years. It is concluded that all patients with osteitis deformans manifesting signs of spinal cord involvement should be subjected to myelography and that a block of the subarachnoid space, if present, should be relieved by adequate laminectomy. Severity of neurological deficit should not be regarded as a reliable guide as to the ultimate benefit from surgery.

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THE TREATMENT OF INTERNAL CAROTID ARTERY ANEURYSMS BY PROXIMAL ARTERIAL LIGATION

A FOLLOW-UP STUDY*

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ANEURYSMS of the internal carotid artery, both in the cavernous and cerebral portions, have been treated in this clinic by proximal arterial ligation in the neck for the past 16 years. We wish to present a clinical review of this experience and to evaluate some of the hemodynamic factors involved in this particular method of treatment. There are 35 patients in the series and, on the basis of a recently completed follow-up study, the present status of all members is known. To date 8 patients have died of various causes and 27 are living.

TECHNIQUE

The diagnosis of internal carotid artery aneurysm was confirmed by arteriography. The procedure was usually performed within a few days of admission, but if the patient's condition demanded prompter action it was done immediately. Bilateral visualization of the anterior part of the circle of Willis was obtained in most cases. In recent years the percutaneous technique has been used initially.

Pre-operative digital occlusion of the common carotid artery was done in all but the critically ill patients. It was compressed 2 minutes 3 times a day at the start and by small increments, over the course of a week or more, increased to 15 minutes 3 times a day. This routine was modified to meet the individual situation. At operation, usually under local anesthesia, the artery to be ligated was occluded under direct vision for no less than 30 minutes. If there were no signs of vascular deficit, the ligature was secured. *In no instance was it necessary to remove a ligature postoperatively.*

The usual site of ligation in patients over 40 years of age was the common carotid. In some instances the internal was subsequently ligated. In younger patients the internal was ligated initially unless there was evidence of existing or impending vascular deficit as determined clinically and by intra-arterial pressure determination at the time of ligation.[2]

To occlude the artery a single ligature of 0 silk was tied over a 1 cm. cuff of deep cervical fascia. Partial ligation was effected by folding the vessel inwards upon itself by means of mattress sutures.[3,4] If cerebral vascular insufficiency impended following the procedure, the patient was placed in a slightly head-down position until the danger was passed. Ordinarily ambulation was begun within 2 weeks.

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RESULTS

Of the 35 patients with internal carotid artery aneurysm, the lesion was located in the cavernous portion in 9 cases and in the cerebral portion in 26 (Table 1).

TABLE 1

Internal carotid artery aneurysms

Cavernous			9
	Dead	1	
	Living	8	
Cerebral			26
	Dead	7	
	Living	19	
Total			35

A. *Cavernous Aneurysms* (Table 2). One patient (A.W.) of this group is now *dead*, succumbing 3 years after ligation to the widespread effects of periarteritis nodosa. Neurologically he improved after ligation. His headaches were relieved, and there was considerable return of function to the extra-ocular muscles. At autopsy, other than findings related to periarteritis nodosa, the left internal carotid artery and the aneurysm thereof were revealed to be small, nonpatent, and filled with scar tissue. The right carotid was about twice normal size; there were no other cerebral vascular lesions.

Eight patients with cavernous aneurysms are *living*. Only 1 person (N.L.) in this group has hypertension. Both patients (R.S. and F.L.) with arteriovenous aneurysms experienced complete cessation of pulsating exophthalmos and bruit after ligation. One patient (D.Y.), blind in the left eye on admission but with no bruit, now almost 16 years later does have a bruit and is suffering from mild headaches which she considers not severe enough to warrant investigation. She is the only patient in this group to report a head pain. Another patient (S.G.) had diminished vision (20/200) in the right eye which improved after common carotid ligation. Following a prolonged period of physical exertion she lost the sight in that eye completely, and subsequent internal ligation failed to improve the situation. Otherwise she is asymptomatic.

All 9 patients with cavernous aneurysms had derangement of the visual apparatus in some manner on admission. Eight patients, 1 of whom was blind, had weakness of the extra-ocular muscles. The other patient in the group had only diminished vision in the right eye on admission and subsequently lost the sight in that eye. Of the 8 patients with extra-ocular muscle deficit, 4 were completely relieved and the other 4 considerably improved. The 4 patients who had trigeminal nerve involvement all experienced clearance of this after ligation.

B. *Aneurysms of the Cerebral Portion of the Internal Carotid Artery* (Table

TABLE 2

Aneurysm of cavernous portion of internal carotid artery

(9 cases)

Patient. Age at Ligation	Symptoms and Findings Before Ligation	Site of Ligation	Survival	Result*
		Died after leaving hospital		
A.W. ♂ 45 yrs.	Headaches, 1 yr. Diplopia, 7 wks. Conscious, alert on adm. B.P. 125/80. N III, IV, VI paresis. Hypalgesia N V(1). Periarteritis nodosa.	Internal, lt.	3 yrs.	Headaches relieved. Eye movements improved. Normal sensation N V(1). Died of periarteritis nodosa. Autopsy.
		Living		
N.L. ♀ 50 yrs.	Transient diplopia, 4 yrs. Headache, ophthalmoplegia, 3 days. Conscious, alert on adm. B.P. 190/108. N III, IV, VI paresis. Hypalgesia N V(2).	Common, rt.	2 5/12 yrs.	Asymptomatic. Slight ptosis.
M.R. ♀ 39 yrs.	Headache, diplopia, 7 mos. Conscious, alert on adm. B.P. 140/96. N VI paresis. Slight ptosis. Dim. corneal reflex.	Common, lt. External, 1 yr. later.	3 4/12 yrs.	Asymptomatic. Neurol. normal.
V.C. ♂ 32 yrs.	Headache, stiff neck, 4 days. Conscious, alert on adm. B.P. 130/80. Slight ptosis. N VI paresis. L.P.: normal pressure; xantho. fluid.	Internal, rt.	6 5/12 yrs.	Asymptomatic. Neurol. normal.
S.G. ♀ 45 yrs.	Dizziness, blurring of vision, 8 mos. Conscious, alert on adm. B.P. 146/96. V.A., rt. 20/200.	Common, rt. Internal, 5 mos. later.	6 11/12 yrs.	Asymptomatic. Blind, rt.
R.S. ♂ 31 yrs.	Throbbing headache after head injury, 2 mos. Diplopia, 4 wks. Conscious, alert on adm. B.P. 130/80. Pulsating exophthalmos. Bruit. N VI paresis. A-V aneurysm.	Internal, lt.	11 5/12 yrs.	Asymptomatic. No bruit. Neurol. normal.

* The results listed in Tables 2 and 3 are the findings observed at the last follow-up examination or are based on information received in response to a questionnaire.

TABLE 2 (*Continued*)

Patient, Age at Ligation	Symptoms and Findings Before Ligation	Site of Ligation	Survival	Result
F.P. ♀ 34 yrs.	Headache, 2 mos. Ptosis, 4 wks. Diplopia, 3 wks. Conscious, alert on adm. B.P. 126/68. N III paresis. Hypalgesia N V(1, 2)	Internal, lt.	11 10/12 yrs.	Asymptomatic. Residual N III paresis.
D.Y. ♀ 33 yrs.	Headaches, many yrs. Failing vision, lt., 6 mos. Diplopia, 1 mo. Conscious, alert on adm. B.P. 138/88. Blind, lt. N III, VI paresis. S.T.S. positive.†	Internal, lt.	15 9/12 yrs.	Headache, not severe. N III and VI paresis improved. Bruit now present.
F.L. ♀ 61 yrs.	Headache, diplopia, "swishing" noise, lt., 3 days. Conscious alert on adm. B.P. 140/80. Ophthalmoparesis, lt. Bruit, pulsating exophthalmos. A-V aneurysm.	Common, lt.	16 8/12 yrs.	Asymptomatic. No bruit. Neurol. normal.

† Serological test for syphilis.

3). There were 2 *operative mortalities*. The patient I.P. presented a desperate situation. Three weeks, and again 4 days before admission, she had severe headaches followed by a generalized seizure and stupor for about 12 hours. There was a similar attack on the day of admission, and she was deeply comatose when we first saw her. Arteriography, done in the operating room, revealed the aneurysm and the common carotid was ligated immediately; ventriculostomy was also done. She died the next day. Autopsy confirmed the location of the lesion and revealed massive intracranial bleeding. The other operative mortality (E.H.) was the only patient in the whole series to have multiple aneurysms, there being lesions of the right internal carotid and right middle cerebral arteries. Ligation in the neck was done first; a direct attack upon the middle cerebral lesion was to have been made subsequently. She died suddenly 12 days after the common carotid ligation. Autopsy revealed the middle cerebral lesion to have ruptured; there had been no bleeding from the internal carotid aneurysm.

Five patients *died after leaving the hospital*. There is reason to believe that 2 fell victim to recurrent intracranial bleeding; the cause of death in 1 patient (A.C.) is not known; the other 2 apparently succumbed to causes other than recurrent bleeding. The lesion in the patient G.B., a hypertensive, was thought to be of the internal carotid at the origin of the posterior com-

TABLE 3

Aneurysm of cerebral portion of internal carotid artery

(26 cases)

Patient. Age at Ligation	Symptoms and Findings Before Ligation	Site of Ligation	Survival	Result
	Operative mortality			
I.P. ♀ 29 yrs.	Episodes severe headache, convulsion, stupor, 3 wks., 4 days, and day of adm. Deeply comatose on adm. B.P. 170/90. L.P.: pressure 600+ mm.; bloody C.S.F.	Common, lt.	1 day	Died. Intracerebral and intraventricular hem. Autopsy.
E.H. ♀ 34 yrs.	Severe, pounding headache, rt., 2 wks. Hemiparesis, lt., 1 wk. Lethargic on adm. B.P. 145/105. Hemiparesis, lt. L.P.: pressure 480 mm., CSF bloody.	Common, rt.	12 days	Died. Hemiparesis improving till sudden death. Intracerebral and intraventricular hem. from rupture of middle cerebral aneurysm. Autopsy.
	Died after leaving hospital			
G.B. ♀ 43 yrs.	Headaches, lt., many yrs. Pain, retro-orbital, 3 wks. N III paresis, 2 wks. Conscious, alert on adm. B.P. 190/120. N III paresis. L.P.: xantho. fluid. Transient aphasia, hemiparesis, after arteriog.	Common, lt.	6 wks.	Headache relieved. N III paresis improved. Returned to work. Died suddenly, ? cerebral hem. No autopsy.
A.P. ♀ 50 yrs.	Headache, loss consciousness, sudden, 7 days. Drowsy, oriented on adm. B.P. 140/80. No lateralizing signs. L.P.: pressure 75 mm.; crenated RBC in CSF.	Common, rt. partial. Total 5 wks. later.	3 mos.	Headache relieved. Hemiparesis, lt. Parkinsonian tremor. Disch. to convalescent home Died of pneumonia. No autopsy.
I.D. ♀ 68 yrs.	Headache, sudden, 3 wks. Headache, ptosis, diplopia, 5 days. Conscious, alert on adm. B.P. 225/90. N III paralysis. Hypalgesia N V(1). L.P.: pressure 140 mm. CSF clear, colorless.	Common, lt.	4 yrs.	Headache relieved. Elevated B.P. persisted. N III paresis. Died of cardiac failure. No autopsy.
A.C. ♀ 75 yrs.	Headache, 5 wks. Diplopia, ptosis, 4 wks. Conscious, alert on adm. B.P. 210/130. N III paresis. Sl. blurring optic disc, rt.	Common, rt.	5 yrs.	Headache relieved. N III paresis improved. Cause of death not known.

TABLE 3 (*Continued*)

Patient. Age at Ligation	Symptoms and Findings Before Ligation	Site of Ligation	Survival	Result
J.P. ♂ 38 yrs.	Headaches, recurrent, 9 wks. Subarachnoid hem. 9 wks. Headache, nausea, diplopia, pain in rt. eye, 4 days. Conscious, alert on adm. B.P. 130/90. N III paresis. L.P. on adm.: clear, colorless CSF.	Common, rt. Internal, 6 wks. later.	9 10/12 yrs.	Asymptomatic except diplopia. N III paresis persisted. B.P. remained normal. Returned to work. Died suddenly, ? cerebral hem. No autopsy.
	Living			
J.M. ♂ 27 yrs.	Headache, severe, sudden, day of adm. Conscious, alert on adm. B.P. 120/75. L.P.: pressure normal, CSF sl. bloody.	Internal, rt.,	4/12 yrs.	Asymptomatic. Neurol. normal. Returned to work.
A.N. ♀ 48 yrs.	Headache, "fainted," 8 days. Hospitalized elsewhere. L.P.: bloody CSF. Trans. to N.H.H. Oriented, drowsy on adm. B.P. 124/65. Subarach. hem. and N III palsy, day after adm. Hemiparesis, hemianopsia, during digital carotid occlusion.	Common, rt., partial. Total 3 wks. later.	7/12 yrs.	No headaches. N III paresis improved. Hemiparesis improved Hemianopsia persists.
C.S. ♀ 52 yrs.	Headache, lt. frontal, 2 wks. Diplopia, 2 days. Unconscious, day of adm. B.P. 120/80. N III paresis, lt. Hemiplegia, rt. Aphasia. L.P.: pressure 240 mm.; bloody CSF.	Common, lt., partial. Total, 4 wks. later.	1 yr.	Speech almost normal. Residual hemiparesis. Sl. N III paresis. B.P. remains normal.
N.T. ♂ 31 yrs.	Headache, confused, vomiting, 1 day. Disoriented, drowsy on adm. B.P. 110/70. Pupil, lt., larger than rt. Devel. aphasia, hemiparesis. L.P.: pressure incr.; CSF bloody. Burr holes: no blood clot.	Common, lt.	1 8/12 yrs.	Speech almost normal. Hemiparesis cleared save for fine finger movements. Has returned to work.
R.C. ♀ 44 yrs.	Headache, sudden, lt., unconscious 30 mins., 2 wks. Hospitalized elsewhere. L.P.: bloody CSF. Trans. to N.H.H. Conscious, alert on adm. B.P. 140/100 Neurol. normal.	Common, lt.	2 9/12 yrs.	Asymptomatic. Neurol. normal.

TABLE 3 (continued)

Patient. Age at Ligation	Symptoms and Findings Before Ligation	Site of Ligation	Survival	Result
D.J. ♀ 52 yrs.	Numbness, lt. index, middle fingers, 2 wks. Headache, stiff neck, 2 days. Conscious, alert on adm. B.P. 122/74. Sl. lower facial paresis, lt. L.P.: pressure 110 mm.; CSF bloody. Transient lt. hemiparesis after arteriog.	Common, rt.	2 10/12 yrs.	Asymptomatic. Neurol. normal.
I.A. ♀ 33 yrs.	Headaches, intermittent, 5 yrs. Headache, severe with vomiting, hemiparesis, 3 days. Semicomatose on adm. B.P. 110/60. Hemiparesis, lt. L.P.: pressure 280 mm.; xantho. CSF. Craniotomy day after adm.: evac. rt. temp. lobe hematoma.	Common, rt. 3 wks. after craniotomy.	3 1/12 yrs.	Asymptomatic. Motor strength equal bilat. Upper quad. field defect, lt. On anticonvulsant medication.
E.C. ♀ 26 yrs.	Headache, generalized, 1 yr. Unconscious, momentary, day before adm. Drowsy, disoriented on adm. B.P. 120/75. Abd. reflex dim., lt. L.P.: pressure 350 mm.; bloody CSF.	Internal, rt.	3 3/12 yrs.	Asymptomatic. Neurol. normal.
V.O. ♀ 47 yrs.	Pain, sudden, lt. eye, 9 wks. Diplopia, ptosis, 6 wks. Conscious, alert on adm. B.P. 140/85. N III paresis, lt.	Common, rt.	3 5/12 yrs.	Residual N III paresis.
E.DiN. ♀ 31 yrs.	Headache, nausea, drowsy, 3 hrs. Conscious, drowsy on adm. B.P. 120/75. Facial weakness, lt. L.P.: pressure 320 mm.; bloody CSF.	Common, rt. Internal 6 days later.	3 8/12 yrs.	Premenstrual "migraine" headache. Neurol. normal.
R.B. ♂ 29 yrs.	Loss consciousness, rt.-sided seizure, 3 hrs. Oriented, drowsy on adm. B.P. 122/80. Sl. facial weakness, rt. L.P.: pressure 500 mm.; bloody CSF.	Internal, rt.	3 11/12 yrs.	Asymptomatic, neurol. normal, until 25 mos. after ligation. Then embolus middle cereb. art. Aphasia, hemiparesis, convulsions. Improving. Anticonvulsant medication
M.M. ♀ 60 yrs.	Headache, dizzy, 7 wks. Loss consciousness, sudden, 10 days. Unconscious next 6 days at another hosp. Trans. to N.H.H. Stuporous on adm. B.P. 150/90. No localizing or lateralizing signs. L.P.: normal pressure; xantho. CSF.	Common, lt.	4 5/12 yrs.	Headaches, generalized, frequent. Neurol. normal.

TABLE 3 (continued)

Patient. Age at Ligation	Symptoms and Findings Before Ligation	Site of Ligation	Survival	Result
A.P. ♀ 38 yrs.	Headache, momentary loss consciousness, 2 wks. Adm. another hosp.; L.P. bloody CSF. Unconscious, ptosis, rt., 5 days. Trans. to N.H.H. Conscious, euphoric on adm. B.P. 125/80. No localizing signs.	Common, rt.	5 1/12 yrs.	Asymptomatic. Neurol. normal.
M.C. ♀ 28 yrs.	Headaches, dull. 2 mos. Sudden occipital pain, nausea, amnesia for the event, 5 wks. Diplopia, 5 wks. Adm. elsewhere; L.P.: bloody CSF. Trans. to N.H.H. Conscious, alert on adm. B.P. 128/78. Neurol. normal.	Internal, lt.	6 7/12 yrs.	Asymptomatic. Neurol. normal.
M.S. ♀ 30 yrs.	Headache, blurred vision, 9 days. Vision improved, headache worse; led to adm. Conscious, alert on adm. B.P. 105/60. Slight hemiparesis, lt. L.P. not done.	Internal, rt.	6 8/12 yrs.	Asymptomatic. Neurol. normal.
M.K. ♀ 38 yrs.	Headache, loss consciousness for 2 wks., 7 yrs. ago. Pain lt. eye, 2 days. Conscious, alert on adm. B.P. 124/70. N III paresis devel. in next 24 hrs. Eye pain stopped by carotid pressure.	Common, lt.	7 8/12 yrs.	Asymptomatic. Sl. ptosis.
E.M. ♀ 28 yrs.	Headaches, severe, 1 wk. "Numb all over," 4 days. Stiff neck, vomiting, day of adm. Conscious, restless on adm. B.P. 120/68. S.T.S. positive; during treatment for this devel. N III paresis. L.P. on adm.: pressure incr.; bloody CSF.	Common, rt.	9 1/12 yrs.	Serology neg. Residual N III paresis.
V.D. ♀ 56 yrs.	Headache, momentary unconsciousness, 1 mo. Ptosis, stiff neck, 1 wk. Hosp. elsewhere; L.P.: bloody CSF. Trans. to N.H.H. Conscious, emotionally labile. B.P. 120/80. N III paresis, lt.	Common, lt.	13 7/12 yrs.	Asymptomatic. Neurol. normal.
J.M. ♂ 23 yrs.	Headaches, intermittent, many yrs. Episode nausea, vomiting, loss consciousness, 3 mos. Similar episode, 2 mos.; hosp. elsewhere; L.P.: bloody CSF. Trans. to N.H.H. Conscious, alert on adm. B.P. 110/70. Neurol. normal.	Internal, rt.	13 7/12 yrs.	Asymptomatic. Neurol. normal.

municating artery. In view of her sudden demise, however, possibly the aneurysm was actually of the communicating artery, and, if it were, the ligation served to increase the blood flow through the vulnerable vessel. The patient A.P. satisfactorily withstood digital compression before operation but displayed contralateral weakness when the common carotid was compressed under direct vision at operation. A partial occlusion was done, therefore. But in spite of this stratagem she had another bleeding episode 2 weeks later and became hemiparetic. Thereupon the arteriogram was repeated and the aneurysm was seen to have enlarged. There was no evidence of an intracerebral hematoma. At that time she withstood complete occlusion of the common carotid without becoming worse. Postoperatively she regained some strength but succumbed later to causes other than intracranial bleeding in a nursing home. The patients I.D. and A.C., both of whom were hypertensives, had considerable improvement in their N III paresis during the time of survival. The patient J.P. was asymptomatic save for a very slight N III paresis and was working regularly until he died suddenly, apparently of a cerebral hemorrhage, 10 years later.

Nineteen patients with aneurysms of the cerebral portion of the internal carotid are *living*. A significant feature of this group is, with 1 exception (M.M.), the absence of hypertension among its members. Certain of them merit discussion. The patient A.N. had a subarachnoid hemorrhage 8 days before admission but, other than being drowsy and having a stiff neck, she presented no focal signs. The day after admission she had another bleeding episode and a N III palsy developed. Arteriography was done immediately. She was to have had a common carotid ligation, but during the interval of occlusion of the vessel under direct vision a left hemiparesis developed. For that reason the arterial wall was folded inwards upon itself, reducing the lumen by about one third. She was also noted to have a left homonymous hemianopsia postoperatively. The hemiparesis and N III weakness gradually improved, and it was possible to complete the common carotid occlusion without reactivating her neurological deficit. At present, although the hemiparesis is improved, the hemianopsia persists. She displayed a transient rise of blood pressure during the phase of cerebral vascular insufficiency.

In contrast to the previous patient, a preliminary partial ligation worked well in the case of C.S. Her lesion was on the left, and she had bled into the temporal lobe. Rather than run the risk of adding vascular insufficiency to an already poor situation, the common carotid ligation was performed in two stages. Her course has been one of steady improvement.

In the patient I.A. the initial arteriogram revealed a large mass in the right temporal lobe but no aneurysm. A craniotomy was performed and a blood clot was removed, still without visualizing an aneurysm. On a postoperative arteriogram, however, the lesion was visualized and the common carotid was subsequently ligated. Her only neurological deficit is a slight quadrantic field defect.

The patient R.B. was asymptomatic for 25 months after ligation. He

then sustained a sudden right hemiparesis and aphasia. This was believed to have been caused by an embolus and no further operative procedures were done. Over the ensuing months there has been some improvement in both the aphasia and hemiparesis.

In this group with aneurysms of the cerebral portion of the internal carotid artery, 12 patients had impairment of the extra-ocular movements prior to ligation. At their most recent follow-up examination, 9 continue to display some N III weakness although in every instance it was improved over the pre-operative status.

DISCUSSION

While the principle of proximal ligation for intracranial aneurysms is still, to a considerable degree, on an empirical basis, we should like to mention some of the known data regarding cerebral circulation and theoretical hydraulics in relation to this problem. The therapeutic objective is simple: to reduce strain on the aneurysm while maintaining adequate circulation to all parts of the brain. Strain upon the aneurysm is equal to the sum of the stresses operating. These include: total hydrostatic pressure in the parent artery, the pulsatile nature of the flow, turbulence, and jet action.

Total hydrostatic pressure is equal to pressure head plus velocity head. Under normal conditions in the circulatory system, with its relatively high pressure and low velocity, the velocity head is relatively unimportant. For example, the theoretical difference of pressure in a needle pointed acutely upstream in the carotid artery versus one pointed downstream is only about 2 per cent. However, the velocity head cannot be ignored, since it increases with the *square* of the velocity. This consideration may be of practical importance in relation to pressure estimations in the internal carotid artery after occlusion of the common carotid. Thus, with an assumed pressure head of 25 mm. Hg following ligation, without change in velocity, there would be an 8 per cent difference in total pressure, depending upon whether the flow were reflux down the internal carotid or collateral from the external carotid. The *higher pressure* would then be found in the *less favorable* condition of internal carotid reflux. Regardless of these factors, there is no reason to doubt that a significant reduction in hydrostatic pressure in the internal carotid occurs distal to ligation.

Returning to the other possible stresses upon the aneurysm, the pulsatile flow may be regarded as having a water-hammer effect within the aneurysm. This pressure wave, the systolic thrust, travels at a much faster rate than the blood, probably about 350 cm./sec. A head of but 10 mm. Hg for example, riding the blood stream at this speed, is capable of a thrust of approximately five times that amount. It is not presently known how much of this systolic thrust is imparted to the inside of the aneurysm. However, there is ample reason to believe that the systolic thrust is very materially reduced in the internal carotid by proximal ligation.

The factor of turbulent flow is difficult to assess. The recent report by

Richardson and Kofman[5] of the presence of intermittent bruits in association with simple intracranial aneurysms suggests that turbulent flow may sometimes occur. However, our knowledge of rheology is not sufficient to evaluate its effect on the aneurysm, if it does occur.

Finally, a few words about jet action in relation to aneurysms. Blood issuing through a small aneurysmal neck at the side of the parent artery carrying an assumed pressure of 100 mm. Hg would have a theoretical velocity of about 500 cm./sec. Actually the jet velocity must be greatly dampened by the blood already in the aneurysmal sac, which must return to the parent artery before more can enter. However, the pulsatile pressure changes in the parent artery would allow ingress and egress of small amounts of blood through the aneurysmal neck. The rapid flow of even small amounts of blood back and forth through a small orifice such as the aneurysmal neck could cause turbulence and thus a bruit, as mentioned above. The theoretical *energy* of the jet varies with the *cube* of the velocity. Furthermore, it has two components, the impulse and the reaction, each of which varies as the square of the velocity, the former tending to drive the aneurysm away from the parent artery, the latter to tear the artery from the aneurysm. The dynamic pressure caused by the action of a jet issuing from an orifice in a vessel is double the hydrostatic pressure on the orifice when closed. These highly significant forces are held in check by the blood within the aneurysmal sac. When a leak occurs they are unleashed. This is the type of force that can drive a hematogenous wedge through the brain and into the ventricular system in a short time. When spontaneous arrest occurs it is probably caused by plugging of the orifice, either from within or without. The effect of proximal ligation upon jet action would be to lower the velocity by reducing the hydrostatic head, the velocity being proportional to the square root of the head.

Returning briefly to the second therapeutic objective, the maintenance of adequate circulation to all parts of the brain. It is apparent from the studies of Shenkin and his co-workers[8] that total cerebral blood flow may be unchanged after ligation of one common carotid. This requires either a rise of mean arterial pressure or a decrease of cerebral vascular resistance. Carotid ligation in the presence of greatly increased resistance, either in the communicating arteries or beyond the circle of Willis, such as may occur with a large intracerebral hematoma, is likely to result in a serious vascular deficit in the homolateral hemisphere. There is need for a less cumbersome method of determining cerebral vascular resistance. In our series this factor was assessed on the basis of the patient's general condition, the clinical response to carotid occlusion and study of the angiograms.

CONCLUSION

When this series was begun there was ample evidence in the literature[7] that internal carotid artery ligation, for conditions other than aneurysms, might be followed by significant morbidity and mortality. Furthermore,

statistics upon the natural course of the disease were not available, and there is still need for data concerning the potential risk of untreated aneurysms in the various intracranial arteries. In order to avoid the possibility that the cure might be worse than the disease, the previously mentioned criteria were set up for the various carotid ligations, and we have had few occasions to depart from them in the ensuing years. An additional factor is the presence of a large intracerebral hematoma, when carotid ligation appears particularly hazardous. Under the conditions of this series, appropriate carotid ligation in the neck has been a reasonably benign procedure and we have not had occasion either to remove a ligature or to resort to trapping a carotid aneurysm. *We do not advocate ligation in the neck for aneurysms of arteries other than the carotid, especially of the communicating arteries.* It is our impression that most of the patients in this series have benefited by the procedure, but the percentage gain in terms of morbidity and mortality can be assessed only in relation to comparable series of untreated cases.[1,6,9]

SUMMARY

A follow-up study of 35 patients with aneurysms of the internal carotid artery revealed that proximal arterial ligation in the neck was a reasonably benign and effective procedure.

While the principle of proximal ligation for intracranial aneurysm is largely on an empirical basis, a study of the stresses operating upon the aneurysm—total hydrostatic pressure in the parent artery, the pulsatile nature of the flow, turbulence, and jet action—tends to indicate that the procedure has a sound theoretical basis.

We are indebted to our colleagues, Drs. Bernard S. Brody and Lycurgus M. Davey, for permission to include their cases.

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TECHNIQUE AND THE DANGERS OF CEREBRAL ANGIOGRAPHY

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DURING the period February 1951–January 1953, the investigation of cerebral angiography was carried out on 430 patients in the Department of Neurological Surgery of the General Hospital, Newcastle-upon-Tyne.

Three deaths and 30 complications resulted from the investigations. Of the complications, 18 were transient and 12 severe and permanent. Hemiplegia or some degree of limb paralysis occurred in every case. In 14 there was also dysphasia and in 10 there was mental confusion or more severe impairment of consciousness.

The object of this paper is to place on record details of the deaths and complications and to enquire why they occurred and how they can be avoided in future.*

DETAILS OF THE INVESTIGATIONS IN THE WHOLE SERIES

Ninety-six bilateral and 334 unilateral investigations were carried out. In 313 instances, 2 injections of 12 cc. of dye for each investigation were made—one for lateral and one for anteroposterior views; in 34 instances, 1 injection of dye only was made—for lateral views; in 83 instances, 3 or more injections for each investigation were necessary to solve special problems or because of technical imperfections.

In 417 instances, the injections were carried out percutaneously; of these, 394 were made under local anaesthesia with 2 deaths and 27 complications and 23 under general anaesthesia with 2 complications. In 13 instances, injections were carried out after the common carotid artery had been exposed; 7 of these operations were performed under local anaesthesia with 1 complication, and 6 were performed under general anaesthesia with 1 death.

The injections of dye were made at room temperature, in quantities of 12 cc. for each shot or view, a 4″ 17-G. needle being used. The speed and force of the injection were determined by strong thumb pressure on the plunger of the syringe. The time taken to empty a syringe was 2 seconds. The lack of control of the force of the injections will be discussed later. Normal saline

* Access to the bibliography on this subject can be gained by reference to the recent publications of Walsh and Smith,[4] Chusid *et al.*,[1] and Ecker.[2]

at body temperature was used to wash the needle through and to keep it clear of blood clot.

THE PROBLEM

There seem to be three possible causes of the complications: (1) The type of case selected for investigation; (2) the irritant nature of the dye; and (3) avoidable or unavoidable faults in surgical technique.

(1) TYPE OF CASE SELECTED FOR INVESTIGATION

Table 1 gives the absolute and percentage occurrence of the permanent and transient complications in each of the 12 groups of the 430 cases in which angiography was carried out.

TABLE 1

Whole Group		Complications			% Complication Occurrence	
Disease	No.	Deaths	Permanent	Transient	Permanent	Transient
Cerebral thrombosis	22	1	1	4	4.5	18.2
Spontaneous subarachnoid haemorrhage	57	—	2	8	3.5	14.03
Nonbleeding saccular aneurysm	37	—	—	—	—	—
Cerebral tumour	59	1	1	3	1.7	5.1
Cerebral angioma	9	—	1	—	11.1	—
Migraine	28	—	1	—	3.6	—
Epilepsy	34	—	—	—	—	—
Head injury: subacute phase	12	—	3	1	25.0	8.3
Cerebral abscess	6	—	—	—	—	—
Atypical facial neuralgia	5	—	—	—	—	—
Degenerative encephalopathy	32	1	3	2	9.4	6.2
Miscellaneous	129	—	—	—	—	—
Totals	430	3	12	18	2.8	4.2

Our experience thus shows that cerebral angiography in cases of cerebral thrombosis is dangerous and that it should be carried out only when a special clinical scientific problem must be solved.

Angiography is also dangerous in the acute and subacute phases of a head injury, but may be used with reasonable safety in cases of chronic subdural haematomata.

Although cautious use should be made of the investigation in cases of large cerebral tumours, it is justifiable if the information gained will lead to better surgery. Mere demonstration, however, of the same information by different methods—pneumography and angiography—is, of course, entirely unjustified.

For the localisation and delineation of aneurysms, whether bleeding or not, cerebral angiography is essential if surgical treatment is contemplated.

In our miscellaneous group of 129 cases, in which clinically there was no evidence of cerebrovascular disease or of cerebral neoplasm, there were no complications. None the less, in spite of this apparent safety when vascular disease or anomaly is absent, we now believe that cerebral angiography in cases of doubtful clinical diagnosis, in the possibility that "something" might be shown by the investigation, should be avoided. On the other hand, we believe that in a few specially selected cases cerebral angiography, even when known to be dangerous, is justifiable in the interests of the advance of knowledge.

(2) THE DYE

Three dyes,* made by two firms, have been tried and, in our experience, no one particular brand of dye was safer or gave better pictures than another. In 141 cases a dye of 42 per cent strength was used and there were 9 complications; in 289 cases a dye of 35 per cent strength was used and there were 21 complications and 3 deaths. We consider it obvious that the lowest strength of dye known to produce pictures of adequate density is the safest.

Autopsy Findings (B.E.T.). Six patients came to postmortem examination within 10 days of the investigation. It was considered that in only 3 instances had the investigation caused or substantially contributed to death.

Case 1. A woman, aged 52 years, was investigated for weakness of the right arm and leg of 3 years' duration. Immediately after angiography, a right hemiplegia developed and she became unconscious, remaining so until her death 48 hours later.

At autopsy, the naked-eye appearances of the brain were those of an acute haemorrhagic infarction of the areas of the left cerebral hemisphere supplied by the anterior and middle cerebral arteries. Microscopical examination confirmed this, but the most striking feature was the large number of hyaline thrombi in the capillaries and arterioles, almost all of which had swollen and necrotic walls, many heavily infiltrated with leucocytes. The appearances strongly suggested that the infarction was caused by an acute necrotising lesion of the small cortical vessels.

In this case death was attributed solely to angiography.

Case 2. A man, aged 41 years, was suffering from a large cerebral astrocytoma. Immediately after angiography a hemiplegia developed and he died 36 hours later.

A small recent haemorrhage had occurred into the tumour, but a large area of cerebral cortex, far from the tumour, was involved in a process similar to, though less severe than, that described in Case 1.

Here it was judged that angiography was the immediate cause of death.

Case 3. A woman, aged 47 years, was investigated for a paresis of the right arm, thought to be of vascular origin. Angiography was followed immediately by a complete hemiplegia. She died 48 hours later.

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* *Uriodone*—Injection of Diodone B.P. containing 35% of Diodone, manufactured by Pharmaceutical Specialties (May & Baker) Ltd. *Arteriodone*—Injection of Diodone B.P. containing 42.5% of Diodone, manufactured by Pharmaceutical Specialties (May & Baker) Ltd. *Pyelosil*—Injection of Diodone B.P. containing 35% of Diodone, manufactured by Glaxo Laboratories Ltd. *Diodone*—The diethanolamine salt of 3:5-di-iodo-4-pyridone-N-acetic acid.

At autopsy, the right middle cerebral artery was thrombosed and part of the hemisphere supplied by that vessel was infarcted.

Here, angiography apparently precipitated a major vascular catastrophe in a patient with a minor vascular lesion.

(3) SURGICAL TECHNIQUE

Surgical difficulties may lead to the necessity of multiple punctures with resulting damage to the vessel walls and to important neighbouring structures.

Bleeding into the Tissues of the Neck. In each of the cases that came to necropsy, there was bleeding into the periarterial tissues; in 3 the bleeding was minimal and closely localised to the site of arterial puncture. In 1 the bleeding was widespread and in 2 blood had trickled into the anterior mediastinum. In no instance was the haemorrhage judged to have contributed to death, either by its quantity or by its pressure on important structures.

Damage to the Walls of the Arteries Subjected to Puncture. The intima on the posterior wall of all the carotid vessels punctured had received one or more lacerations up to 3 mm. in length. In one instance there was a haemorrhage in the media, 1 cm. long and 5 mm. broad. In one instance a mound of friable blood clot about 5 mm. across, projecting into the lumen of the vessel from one of the lacerations in the intima, was an obvious source for possible cerebral emboli. Indeed, all these lacerations were possible sources for delayed cerebral emboli.

Speed and Force of the Injection. As we felt that damage to the cerebral capillaries might result from the force of the injection, experiments in 2 selected cases were carried out, to prove this point. With a bone flap raised and the cortex about the Sylvian fissure under direct vision, injections were made into the common carotid artery on the same side, using our usual technique for cerebral angiography. Cinematographic records in colour were also made during the experiments.

On those occasions when either dye or saline was injected rapidly and under pressure, dramatic colour changes in the cerebral cortical background were observed. The cortex immediately blushed a vivid red, then blanched, blushed again and returned to its normal colour, the cycle of colour changes taking a few seconds only. When dye or saline in the same quantities were injected slowly, either at room or body temperature, no colour changes were observed. The impression given in the colour changes was that of a column of fluid flowing along the cerebral vessels driving a column of blood before it.

In order to analyse this impression, a further 12 cc. of normal saline, coloured with 1 cc. 5 per cent methylene blue, were injected under pressure. The colour changes were those of blushing, blueness, blushing and return to normal colour. When 12 cc. of citrated blood were injected under pressure, no obvious colour changes were detected.

From these experiments, it is clear that it would be possible, by forcible

injection of fluid through a syringe, to drive out the blood from the capillaries of the brain for a sufficient length of time to cause severe ischaemia.

Saline. Saline solutions, unless carefully prepared and made strictly isotonic, will act as irritants. From our experiments it will be seen that if too much saline is used to keep the needle free from clot, blood will be washed out of the cerebral capillaries and ischaemia will result. Prolonged ischaemia from any cause will, of course, lead to irreversible damage to the neurones.

COMPLICATIONS IN RELATION TO THE NUMBER OF INJECTIONS

Complications developed

(a) in 3 instances after the 1st injection of dye; the total number of patients subjected to 1 injection only was 34.
(b) in 11 instances after the 2nd injection of dye; the total number of patients subjected to 2 injections was 217.
(c) in 9 instances after the 3rd injection of dye; the total number of patients subjected to 3 injections was 83.
(d) in 10 instances after the 4th injection of dye; the total number of patients subjected to 4 injections was 96.

TIME RELATIONSHIP OF THE COMPLICATIONS

Of the 12 permanent complications, the development of the symptoms occurred as follows:

(a) in 8 instances immediately.
(b) in 1 instance within 1 hour.
(c) in 1 instance within 6 hours.
(d) in 2 instances within 24 hours.

Of the 18 transient complications, the development of the symptoms occurred as follows:

(a) in 13 instances immediately.
(b) in 2 instances within 1 hour.
(c) in 3 instances within 24 hours.

PRE-OPERATIVE AND POSTOPERATIVE STELLATE GANGLION BLOCKS

In the whole series, pre-operative injection of the stellate ganglion with 1 per cent Procaine to prevent spasm of the vessels was carried out in 20 cases only; in 2 of these, complications developed. In all cases the periarterial tissues were generously infiltrated with Procaine.

In 13 cases of the complication group a postoperative stellate ganglion block was performed; in 4 cases only was there any significant relief of symptoms and in each instance the relief was dramatic.

LATE RESULTS

Of the 12 patients with permanent complications, 3 eventually died of their original disease. Of the remaining 9 patients, their disability on discharge from hospital was as follows: 4 had weakness of an upper limb, 1 had weakness of one hand, 2 had a hemiparesis, 2 had a dysphasia, 1 also having weakness of an upper limb.

DISCUSSION

The incidence of complications in our series seems to have been higher than that observed by others, but many clinics now realise that the investigation of cerebral angiography is not so safe as it was once thought to be. One of us (G.F.R.) encountered his first complication in the investigation about 20 years ago:

"Once when dissecting in the neck under local anaesthesia, in order to carry out cerebral angiography, the internal and external carotid arteries went into extreme spasm. Immediately severe pains were complained of in the same side of the head and face, and the patient slowly developed a contralateral hemiplegia, which equally slowly receded."[3]

There are many possible causes for complications, such as haemorrhagic capillary infarction because of the toxic nature of the dye; spasm of the large vessels of the neck; spasm of the vessels of the brain; washing out of the blood from the brain by an injected column of fluid; and cerebral emboli.

ADVICE

In order to minimize complications, we advise the following:

(1) Careful selection of cases.

(2) Restriction of the number of radiological views, so that if lateral views give the required information no further films are taken.

(3) The designing of a new syringe by which a minimal force of injection that gives adequate concentration of the dye only is used.

(4) Discontinuance of the investigation when difficulty is experienced in entering the artery.

(5) The preparation of a more satisfactory form of dye that can be injected slowly and produce an adequate concentration for radiography.

(6) The careful preparation of normal saline and use of a minimal quantity to keep the needle clear.

Despite its dangers, we consider that cerebral angiography is one of the great advances in neurology, and we hope our experiences will help others in the better selection of cases for it.

SUMMARY

Of 430 patients subjected to cerebral angiography, 33 had postoperative hemiparesis, associated with neural dysfunction; 3 of these patients died as a direct result of the investigation. The causes of complications are reviewed and suggestions made on their avoidance, particularly with reference to the selection of cases in which the investigation should be carried out.

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PARAPLEGIA CAUSED BY SPONTANEOUS SPINAL EPIDURAL HEMORRHAGE*

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PARAPLEGIA resulting from spontaneous spinal epidural hemorrhage is rarely encountered. Review of the literature from the first reported case of Bain[1] in 1897 to the case of Svien, Adson, and Dodge[9] in 1950 reveals a total of 12 cases. Ver Brugghen[10] reviewed the first 7 of these and pointed out the paucity of information on spinal epidural hemorrhage to be found in current textbooks. He emphasized the importance of prompt recognition of spinal epidural hemorrhage and recorded the only case in which early surgical removal of an epidural blood clot from the cervical region resulted in prompt recovery from quadriparesis. Kaplan and Denker[6] in 1949 summarized the previous literature and reported 2 cases of their own in which the lesions were treated surgically. It was their belief that spinal epidural bleeding was of venous origin caused by a rupture in the weak, vascular wall of a pre-existing abnormal epidural venous plexus. They admitted the lack of convincing pathological evidence. Chavany *et al.*[2] added another case of lumbar epidural hemorrhage. Svien and co-workers[9] called attention to the clinical similarity of epidural hemorrhage and the lumbar disk syndrome. Our own recent experiences corroborate this.

Spontaneous spinal epidural hemorrhage is poorly understood. We have little doubt that undiagnosed cases occur frequently. Neurologic findings may be transient and bizarre, depending upon the amount of hemorrhage. When the hemorrhage is small, little or no neurologic changes ensue, and the usual symptomatic treatment of back pain suffices. There is presumptive evidence that recurrent episodes of back pain may result from repeated spinal epidural hemorrhage. When massive hemorrhage occurs, marked motor and sensory impairment follow. Only by recognition of the pathology and prompt surgical removal of the clot can satisfactory neurologic recovery be anticipated. Recently, we have had the opportunity to treat 4 patients with massive spontaneous spinal epidural hemorrhage. Our purpose of reporting these cases is to re-emphasize this grave neurologic lesion and to

* The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration. Case 4 was reviewed in the Veterans Administration and published with the approval of the Chief Medical Director.

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encourage others to record their experiences. It is our belief that only by the reporting of individual cases will a better comprehension of the etiology and treatment occur. Furthermore, we wish to emphasize that spontaneous spinal epidural hemorrhage is apparently more common than we have heretofore believed.

Case 1. #63810. F.G.G., a 24-year-old white apprentice printer, was seen by one of us (A.C.J.) Dec. 6, 1951. According to his physician, Dr. T. F. Walker, Jr., of Great Falls, Montana, he had been in good health until the previous evening when he experienced severe, stabbing pain in the upper thoracic spine about 1 hour after heavy lifting. Flexion of the trunk aggravated the condition. Later during the night, pain radiated anteriorly in a distribution more or less corresponding to the 5th thoracic dermatome bilaterally. An analgesic was given. By morning he experienced numbness and weakness of the legs, progressing to a complete paraplegia with a transverse sensory level in the upper thoracic region.

Examination, 18 hours after onset of symptoms. He was a well-developed and nourished male, alert, oriented, and cooperative. Normal motor power was present in the upper extremities. The lower extremities were flaccid, although the deep reflexes were present, equal, and active to slightly increased. Voluntary motion had been affected in the right lower extremity initially, and on this side there was a Babinski sign. On the left this sign was absent. Deep sensation on pinching the small toe was still present on the right side but absent on the left. There was a complete loss of superficial sensation as high as the 5th thoracic dermatome on the right and the 6th on the left.

The clinical impression was that this patient had an acute transverse myelopathy, apparently originating from the right side of the spinal cord at about T5 cord level (T3 vertebral level). The two most likely possibilities appeared to be an acute extrusion of the nucleus pulposus or a spinal epidural hematoma. In either event immediate decompressive laminectomy was indicated. Because of the rarity of herniation of the nucleus pulposus at the level indicated in this case, some doubt was cast on this possibility, though it was also recognized that a spinal epidural hematoma is a very rare occurrence, at least in the absence of known specific trauma.

Roentgenograms of the upper dorsal and lower cervical spine revealed no evidence of fracture, dislocation, or erosion.

Spinal puncture at the 4th lumbar level yielded normal-appearing fluid. There was a complete block to the Queckenstedt test. One cc. of pantopaque (ethyl iodophenylundecylate) was inserted into the lumbar sac. With the fluoroscopic table tilted down to approximately 60° from the horizontal, there was evidence of a complete block with a concave "tumor cap" type of defect corresponding with the lower level of the 3rd thoracic vertebral body. A small specimen of CSF contained 50 mg. per cent of protein and no cells.

Operation. The patient was immediately taken to surgery, where under endotracheal anesthesia a complete laminectomy of the 2nd through the 4th thoracic vertebrae was carried out (A.C.J.). As soon as the removal of the laminae was begun a moderately large, dark, "currant jelly" epidural clot was disclosed lying dorsal over the dural sac and markedly compressing it to a depth of about 1 cm. at its deepest portion. After removing a portion of this for pathologic study, the remaining hematoma was completely removed by suction and irrigation. Complete relief of the block was immediately evident by the development of normal pulsations of the dura

mater. Intradural exploration disclosed no gross abnormality and a #8 F. catheter could be readily passed 10 to 15 cm. in either caudal or cephalic direction without encountering an obstruction. The dura mater was closed tightly with a continuous silk suture and the laminal defect was filled with Gelfoam to obliterate the dead space.

Course. There was rapid and progressive neurologic recovery. On the 1st postoperative day a flicker of voluntary contraction of the left quadriceps with extensor movements at the knee joint was noted. Good deep and superficial sensation and sensory localization were present in both legs. On the 2nd day there was movement of the toes of the left foot and physiotherapy was begun. Voluntary contractions of both flexors and extensors at the left hip, knee, and ankle were present on the 3rd day. Slight extensor movements were present at the right knee, ankle, and toes. By the 5th postoperative day he was up in a chair with continued progressive improvement in motor power in both lower extremities, and on the 6th day nearly normal motor power was present in the left lower extremity and only slightly reduced motor power in the right extremity. During the 1st postoperative week, voluntary emptying of the bladder was not possible, and an indwelling catheter was used most of the time. Because of the high urinary residuals of as much as 800 cc., he was put on a regimen of Prostigmine methylsulfate 0.0005 (1/120 gr.) with Doryl (carbaminoylcholine chloride) 0.00025 (1/260 gr.) subcutaneously before attempting to void. Residual urine steadily decreased.

Subsequent follow-up revealed continued recovery. On Feb. 19, 1952, he was practically symptom free, although he stated his legs felt "a little heavy" when running. He was free from any subjective limitation with usual physical activity. The only objective neurologic findings consisted of a slight briskness in the knee and ankle reflexes, particularly the latter, and these were somewhat greater on the left side. There was no ankle clonus, and an inconstant and equivocal Babinski sign was obtained on the right side only. On re-examination on Oct. 23, 1952, he was asymptomatic and neurologically normal.

Summary. An acute onset of severe back pain followed by progressive loss of function of lower extremities was found to be caused by an expanding epidural hemorrhage. Evacuation by laminectomy resulted in prompt recovery.

Case 2. #57872. S.W.R., a 79-year-old white male, was admitted to the Urologic Service of Dr. T. D. Moore at the Baptist Memorial Hospital, Memphis on Mar. 11, 1950, complaining of paralysis of the lower extremities, urinary retention, and incontinence of bowel. For the previous 2 years the patient had had urinary difficulty manifested by slowing of the stream and straining. Nocturia occurred 2 to 4 times. A few weeks before admission he had had left sciatic pain after lifting heavy lumber. This pain gradually disappeared. One week prior to admission, while standing and playing with his grandchild, he experienced a sudden onset of excruciating pain in the right hip which spread to the left hip and down the posterior aspect of both lower extremities to the feet. Within a few moments both lower extremities became weak, and numbness of the feet and legs occurred. By holding to a wall, he was able to support himself until help arrived a few moments later. He complained of numbness and tingling of both lower extremities. Voluntary motion of the extremities was lost. Urinary retention and rectal incontinence developed.

He was hospitalized in another city for 8 days where he was treated with bed rest, and an indwelling urethral catheter was inserted. Low back and lower extremity

pain was so severe that the administration of opiates was necessary. Three days after the onset of his illness slight movement returned to the left extremity, and a few days later in the right. It was because of his urinary difficulties, however, that he was transferred to the Baptist Memorial Hospital. We were asked to see him in consultation shortly after admission. Despite the recent administration of a sedative, he complained of severe back and lower extremity pain, especially when moved.

Examination. The patient was obese and in obvious pain. His temperature was 99°F., pulse 90, respiratory rate 18, and B.P. 136/84. Heart sounds were normal and the lungs were clear.

There were no abnormalities of the upper extremities. Both lower extremities were markedly weak. On the right voluntary plantar flexion of the foot was impossible, but all other movements of the extremity could be performed weakly. All muscle groups on the left were functioning, but strength was greatly diminished. There were hypesthesia and hypalgesia over L5-S1 dermatomes bilaterally. Position sense and vibratory sense were normal. Pressure over the sciatic nerves caused severe pain. The Lasèque sign was positive at 150° bilaterally. Compression of the veins of the neck produced low back and extremity pain. The patellar and Achilles reflexes were absent on the right. The left patellar reflex was diminished, the Achilles reflex was absent. Cremasteric reflexes on the right were diminished, on the left absent. Abdominal reflexes were normal. There was marked spasm of the lumbar paravertebral muscles and the lumbar lordotic curve was decreased. Pressure over the 4th lumbar spine produced intense bilateral sciatic pain. There was an indwelling urethral catheter. The anus appeared patulous. On digital examination, slight anal sphincter tone remained. There was a benign enlargement of the prostate to grade 2 plus.

The blood was normal. The urine was normal except for the presence of 1-10 RBC and WBC/low power field. Blood non-protein nitrogen was 42.8 mg. per cent. X-rays of the lumbosacral spine revealed a slight scoliosis to the left, moderate decalcification, and moderate osteoarthritic changes. No definite loss of curve or intervertebral space narrowing were noted.

It was our belief that the patient most probably had suffered an extrusion of a lower lumbar intervertebral disk. However, we felt that lumbar myelography was advisable because of the unusual clinical picture. This was at first refused by the patient. Three days later the procedure was permitted. The spinal puncture, done at the 5th lumbar level, revealed light-yellow CSF under 140 mm. water pressure. It contained 17 lymphocytes and 1 segmented granulocyte. Total protein was 194 mg. per cent. Kline and Queckenstedt tests were negative.

Myelography, using 3 cc. of Pantopaque, disclosed a streaking of the column of oil at the 3rd and 4th lumbar levels with almost complete block of the subarachnoid space at the 2nd lumbar space, suggesting an extradural lesion.

Operation. On Mar. 15, 1950, using local anesthesia, a laminectomy was done from L2 to L4 (E.C.S.). Upon opening the ligamentum flavum, a small quantity of black blood exuded from the epidural space. A well-organized hematoma was exposed, measuring approximately 12 cm. in length and 10 to 12 mm. in thickness. After removal of the clot at the 2nd lumbar level, the dural sac was found to be collapsed. The clot extended anteriorly so that the lumbar sac was completely encircled. The clot was removed by suction and cup forceps. On opening the dura mater, the arachnoid was found to be light yellow in color, slightly thickened and adherent to nerve roots which appeared red and inflamed. By lowering the head of the operating

table, globules of pantopaque could be seen to collect beneath the arachnoid at the 2nd lumbar level, where adherent arachnoid obstructed its cephalad flow. The arachnoid was opened and jugular compression produced a prompt flow of cerebrospinal fluid into the wound. The cause of the hemorrhage was not found. The dura mater was closed tightly with interrupted silk sutures. Routine closure of the wound was carried out. A small Penrose drain was left beneath the paravertebral muscles for 24 hours.

Pathological diagnosis of the tissue removed: organized blood clot.

Course. Motor power returned rapidly over a period of 10 days. Rectal sphincter tone was regained over the same period.

Suprapubic cystotomy was performed on Mar. 27, 1950, and he was discharged to his home 2 days later. He was re-admitted to the hospital ambulatory on June 12, 1950, for transurethral prostatectomy.

When seen Aug. 1, 1950, he complained of slight paresthesias of the right leg and left foot. He denied any pain. Some urinary incontinence from previous prostatic surgery remained. Rectal tone was normal. Both Achilles and patellar reflexes on the right were absent. On the left these were diminished. Straight leg raising and compression of the veins of the neck caused no discomfort. Muscle power in the extremities was normal. There were no abnormalities of sensation.

Summary. Severe low back and lower extremity pain in an elderly male was followed by immediate complete paraplegia. Over a period of several days slight muscle function returned to both extremities. Bowel and bladder incontinence persisted. Operative removal of an epidural hemorrhage resulted in prompt return of normal function.

Case 3. Hospital #186-725. F.D., a white female, aged 56 years, was seen on Sept. 7, 1952, in Le Mars, Iowa, at the request of Dr. F. C. Bendixon. On Sept. 6, 1952, as the patient climbed into an automobile and bent her head to enter the back door, she had experienced a sudden pain in the left flank and slight backache. During the next 15 minutes the pain became excruciating and radiated across the back into both flanks. Examination by a physician revealed only slight left upper abdominal tenderness. Morphine, gr. 3/8, was administered and all pain stopped. There was normal voluntary motion in all extremities. After the doctor left, she noticed tingling sensations in both feet, accompanied by weakness of the extremities. This gradually increased so that in 3 hours there was complete paralysis of both lower extremities. Later in the evening the patient was hospitalized. No change occurred during the night. Lumbar puncture the following morning revealed uniformly blood-tinged fluid that could be obtained only by aspiration.

Neurologic Examination, about 24 hours after the onset. The patient was mentally clear. Acute flexion of the neck with coughing produced aching pain in the flank, but the Naffziger sign was absent. The deep reflexes were normal and equal in the upper extremities. Reflexes were absent in the lower extremities. There was no plantar response bilaterally. The abdominal reflexes were absent. Complete loss of pain, temperature, vibratory and position sense to the 10th thoracic dermatome was found. Straight leg raising on the right produced backache at 115° and was normal on the left. Tenderness was present on pressure over the 12th dorsal spine and caused aching sensation into the left flank and both lower extremities. There was slight tenderness in the left upper quadrant of the abdomen, but no rebound pain was present. The dorsal pedal and femoral pulses were normal, and there was no edema of the lower extremities.

It was learned that in 1950 the patient had had a sudden numbness of the right face and extremities without headache, nausea, or unconsciousness. Recovery was complete except for a slight residual numbness in the right leg. The B.P. averaged 160 to 180 systolic after that time. No hemorrhagic tendency had ever occurred, although she bruised easily. A history was also obtained of occasional pain in the left flank during the last 6 to 8 months. This was increased by hunger and relieved by eating, lasting not over 15 to 30 minutes.

The patient was transferred to St. Joseph Mercy Hospital, Sioux City, Iowa, where myelography was done. The Queckenstedt test revealed a complete subarachnoid block, and 2 cc. of Pantopaque introduced at the 3rd lumbar level flowed cephalad only to the upper border of the 1st lumbar vertebra. Roentgenograms of the chest and spine were normal. Blood studies, including prothrombin time and hematocrit determination, revealed no abnormalities.

Operation. Under general anesthesia, laminectomy (C.A.B.) of T10, T11, and T12 was done 5 hours after initial examination (29 hours after onset of symptoms). This revealed an acute epidural clot extending over the entire length of the exposure. Its approximate length was 7 cm. and it covered the entire dorsal half of the dura mater. This was similar to that in Case 1. The hematoma was easily removed. There was slight epidural bleeding which was controlled by coagulation and oxycel gauze. The dura mater was opened without opening the arachnoid, and normal pulsations were seen. A #10 catheter could be run in the subdural space for a distance of 10 cm. cephalad and caudad without obstruction. The dura mater was closed by a continuous silk suture.

Course. The patient made slow but progressive improvement. There was some motion of the toes of both feet within 12 hours, but she did not void spontaneously for 1 month. A spinal puncture on Sept. 24, 1952 revealed a pressure of 130 mm. Spinal fluid dynamics were normal.

At the time of hospital discharge on Oct. 11, 1952, there was an estimated 50 per cent function in all muscles of the left lower extremity and 25 per cent in the right lower extremity. Epicritic sense was present to the knees in both legs and protopathic sense in the legs and feet.

Three months after surgery the patient was able to walk freely with a stroller and could take 8 to 10 steps alone. She was free of pain and had normal function of bowel and urinary bladder.

Summary. Several hours after the onset of severe back pain, paraplegia occurred. Prompt removal of a low thoracic epidural hematoma was followed by progressive return of neurologic function.

Case 4. C.M.F., a 34-year-old white male, was admitted to the Veterans Administration Hospital, Fort Howard, Maryland, on Aug. 14, 1952. In 1938, 14 years previously, while walking he had been suddenly seized with sharp, stabbing pain in the lumbar region. There was no radiation of pain and it vanished after 1 week of bed rest. Two years later, while lifting, he again had a sudden onset of sharp, stabbing pain in the lumbar region. Following 2 weeks' bed rest the pain subsided. In 1940 low back pain returned while riding in a vehicle. On this occasion the pain was more severe than before and lasted 10 days. Approximately 1 year later, while standing, there was still another abrupt onset of sharp, stabbing pain—worse than ever before—in the low back. Despite hospitalization, the pain remained severe for 3 weeks. Since the initial attack of pain in 1938. he had been bothered by low backache when he began to straighten up after bending over or if he stood in one position for a pro-

longed period. He found that this aching pain was relieved by assuming a squatting position or by bending over as if to touch his toes. He stated that during the 4 years preceding his present hospital admission he had been drinking on an average one pint of whiskey per day.

At 2:30 A.M. on Aug. 14, 1952, the patient awakened with severe pain in his low back which radiated to his lower abdomen and to the posterior aspect of both hips, associated with numbness along the lateral aspect of his thighs and legs. The family physician gave an injection of morphine which relieved the pain; 15 minutes later the patient tried to get out of bed. His legs gave way under him and he fell. Although he was able to walk a few minutes later, pain persisted, and he was hospitalized.

Examination. On admission he was awake, aware, alert, and cooperative. His temperature was 98.6°F., pulse 72, respiratory rate 20 and B.P. 130/70. There was diffuse tenderness over the lumbar spine with paravertebral muscle spasm on the right side. Motor power appeared normal in the lower limbs. At that time pain was limited to the low back and right lower limb. Straight leg raising was painful at 140° on the right with reference of pain to the low back and right hip. Except for a decreased Achilles reflex on the right, the deep reflexes were normal and the plantar responses were flexor.

Urinalysis on August 15 showed 3+ albumin, 6–8 WBC/hpf and a few finely granular casts. On August 19 hb. was 13. 2 gm. (91 per cent), and WBC 13,600 with 85 per cent polymorphs, 12 per cent lymphocytes, 1 per cent monocytes, and 2 per cent eosinophiles. The urea nitrogen was 25 mg. per cent, blood sugar 80 mg. per cent, and the Eagle flocculation was negative. Roentgenograms of the chest and lumbar spine showed no abnormality except an old compression fracture of the body of the 3rd lumbar vertebra. The patient later stated that he had been told of this roentgenologic finding in 1943.

Course. Two days after admission he complained of inability to move his lower limbs and inability to void. He was catheterized and 1150 cc. urine were obtained. Later the same evening he became confused and disoriented. A psychiatrist reported that he was having delusions and hallucinations which centered around his being captured and his wife and child being dead. He was moved into a locked ward and heavily sedated. Three days later he became oriented and was noted to have a flaccid paraplegia. He was seen in neurosurgical consultation on Aug. 24, 1952.

Neurologic Examination. The patient was well oriented and cooperative. Cranial nerves and upper limbs appeared normal. There was a flaccid paralysis of both lower limbs with absent patellar and Achilles reflexes bilaterally. Plantar stimulation on either side evoked no response other than a slow plantar flexion of the great toe associated with some flaring and some flexion of the lesser toes on the stimulated side. There was a sensory level at L1 below which all modalities of sensation were absent. Throughout the lower thoracic and upper lumbar regions, there was diffuse tenderness. When the neck was flexed or when the extended lower limbs were elevated, he complained of severe pain in the lower dorsal region radiating around to the anterior aspect of the lower abdomen. Compression of the jugular veins did not cause back or leg pain.

Lumbar puncture showed a complete block on bilateral jugular compression. The CSF was clear and xanthochromic, with a protein of 196 mg. per cent. There were no cells, and the spinal fluid sugar was 66 mg. per cent.

Operation. Under general anesthesia immediate laminectomy (W.H.M.) was carried out from T9 through L1, exposing a sausage-shaped mass lesion which lay

on the dorsal and right lateral aspects of the dural sac, terminating at the upper and lower ends of the operative field. The dorsal and lateral aspects of the lesion appeared to be encapsulated, whereas medially and anteriorly, the major portion of the mass consisted of black, clotted blood. The hematoma was removed in its entirety and the wound was closed. The dura mater appeared normal and was not opened.

Pathological Report. The tissue removed consisted of blood clot which showed varying amounts of fibroblastic organization. Practically no iron-like pigment was recognized. There were abundant fibroblasts and many capillary-like vessels of varying sizes. Some areas of hemorrhage appeared older than others and it appeared that hemorrhage had occurred in granulation tissue, organizing a previous hemorrhage. No neoplastic cells were seen. Histological examination of bone removed at time of laminectomy showed normal-appearing, blood-forming elements.

In light of the pathologic findings, further blood studies were carried out on Sept. 4, 1952. Hb. was 12.7 gm. (88 per cent), RBC 4,790,000, hematocrit 46, WBC 10,900 with 76 per cent polymorphs, 22 per cent lymphocytes, and 2 per cent eosinophiles, and platelets 384,000. The bleeding time was 1 minute, coagulation time 5 minutes, and the prothrombin time was 16 seconds with control 14.6 seconds.

Course. Immediately following operation, the patient was relieved of pain and from the subjective numbness in the back and lower abdomen. There was no return of motor power, and he remained incontinent of urine and feces. Two days later his sensory level had dropped to L2 and all modalities of sensation were absent below that level. Deep reflexes remained absent and since operation there had been no response to plantar stimulation on the left. Until Aug. 30, 1952, plantar stimulation on the right evoked a slow plantar flexion of the great toe, associated with some flaring and flexion of the lesser toes.

The patient is now up and about in a wheelchair and has been transferred to the rehabilitation service.

Summary. Over a period of 14 years, this 34-year-old man had recurrent backache. On 5 occasions severe back pain had confined him to bed, and he was twice hospitalized because of the pain. During the last hospitalization, a psychotic episode occurred which lasted 3 days. It was then noted he was paraplegic. Operation was carried out 5 days later, and although he was improved by surgery, paraplegia persisted. Had operative removal of the epidural hemorrhage been carried out within a short time after the appearance of extremity weakness or paralysis, a more nearly complete neurologic recovery would undoubtedly have resulted. Microscopic sections of the tissue removed revealed it to be composed of both old, well-organized clot and recent hemorrhage.

DISCUSSION

In a review of the literature[3–5,7,8,11] spontaneous epidural hemorrhage was found to occur at any age from 20 months to 75 years. With but one exception it occurred in the adult age group. There was no sex predominance. All patients had a history of trauma with the exception of a 20-month-old infant, in whom infection was present. It is noteworthy that in most cases in which trauma occurred, it was mild. Straining and twisting efforts seemed particularly prevalent. Severe neurologic impairment occurred in 20 minutes to several weeks after trauma. There were 3 lesions of the cervical

region: 2 confined to the cervical levels, the other extending from C3 to the lumbar area. There were 4 lesions of the upper dorsal region, and 2 of the lower dorsal region, 1 of which extended to the 1st lumbar level. In 3 cases the lesions were confined to the lumbar region. Nine of these patients were treated surgically. Of these, 5 made almost complete recovery—1 lesion was in the cervical, 2 were in the dorsal, and 2 in the lumbar regions. Three made no neurologic recovery, and 1 patient expired 2 weeks postoperatively. To this group of operated cases, we add our own 4.

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CONTROLLED HYPOTENSION AT INTRACRANIAL OPERATIONS

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DURING the past few years various methods have been introduced for the purpose of reducing bleeding during surgical procedures by means of induced and controlled hypotension.

The ganglion-blocking agent, hexamethonium, was introduced at the Neurosurgery Department of Serafimerlasarettet in the fall of 1951 for use at intracranial operations. After one year's experience with over 70 patients in whom this method has been used at operation, our technic is as follows.

TECHNIC

Routine pre-operative examination is made with special attention to blood changes (anemia), cardiac history, state and function, electrocardiographic changes and renal changes.

Anemia is rectified, if possible, by pre-operative transfusions.

Premedication is usually morphine and scopolamine in individually adapted doses. Any fall in blood pressure following premedication is noted.

The routine method of anesthesia is by insufflation through a tube with a caliber as large as possible so as to ensure the greatest possible interchange of gases.

Intravenous drip of physiologic saline solution or glucose solution is given slowly from the start of the operation, though at a sufficient rate to ensure free passage through the cannula or needle. The saline and glucose flasks must be rapidly exchangeable for the administration of pre-operatively cross-tested blood, Dextran or the like.

The operating table is horizontal at the beginning of the operation. When it is desired to reduce the blood pressure, an intravenous injection is given of an individually adapted, though always relatively small dose of some hexamethonium compound, diluted with physiologic saline solution so that 1 cc. is equivalent to 10 mg. The initial dose is usually 10–20 mg.

The effect is checked for 3 minutes. If the required fall has not occurred, the same dose is repeated. If the blood pressure still has not dropped sufficiently after 50 mg., the head of the operating table is raised.

By raising and lowering the head of the table the systolic pressure is adjusted to the level required, under 80 mm. but over 50–60 mm. Hg.

In the event of an excessive drop in blood pressure, the head of the table is lowered and blood is administered in rapid drips.

Should hypotension be required over a longer period than 30 minutes, it is usually

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necessary to administer further hexamethonium in half of the initial effectual dose.

For the duration of hypotension a liberal supply of oxygen and decreased amounts of anesthetic are ensured.

Any loss of blood is immediately made good as far as possible by intravenous transfusion through the drip. Apparatus for intra-arterial transfusion is kept close at hand, facilitating transfusion in the centripetal direction, after rapid exposure of the radial artery, if the patient goes into a threatening state of shock.

When the pressure is to be raised again, this is done progressively by gradually lowering the head of the table. The pressure should not be less than 90–100 mm. Hg at conclusion of the operation. If so, we lower the head of the table slightly below the horizontal plane, this usually bringing about the required rise in blood pressure after only a few minutes. Only in exceptional cases is it necessary to resort to vasopressor agents, Oxedrin or the like. During the postoperative phase the blood pressure is permitted to rise spontaneously to the patient's normal level.

Meticulous supervision is required both at operation and during the immediate postoperative phase. The operative field must be observed, bleeding and changes in color noted, the peripheral circulation watched, the pulse continually palpated and the respiration carefully checked, since respiratory obstruction occurs more readily in hypotension.

Postoperatively the pulse and blood pressure are recorded every 5 minutes for 2 hours or until the blood pressure is stabilized, and thereafter they are checked every 15 minutes.

Postoperative management of the patient is routine, with checks of the blood values, electrocardiograms, urine tests, etc.

INDICATIONS

From the present series one may distinguish three main indications for controlled hypotension at intracranial operations.

(1) *Arterial aneurysms.* In such cases the blood pressure is not reduced until the surgeon approaches the aneurysm, and at its exposure and ligation. With a fall in blood pressure the tension in the aneurysmal sac decreases and the risk of rupture is substantially lessened. Exposure can be done more readily, and if the aneurysm should rupture, the bleeding will naturally be easier to overcome if hypotension is present. At operations for intracranial aneurysms the blood pressure need be reduced below 70 mm. Hg only for a few minutes.

(2) *Highly vascular tumors and malformations*, such as meningiomas and arteriovenous aneurysms. It was an early experience in neurosurgery that at operations on highly vascular intracranial tumors the surgeon made but little progress in the early stages of the operation because of bleeding from the tumor and surrounding tissues. When the blood pressure commenced to fall after a certain loss of blood, removal of the tumor proceeded more rapidly. This observation prompted Gardner (1946) to develop his method involving arteriotomy at the beginning of the operation. The same results can be obtained with ganglion-blocking agents, administered intravenously, and induced hypotension. In such cases the operation can be completed more rapidly and with less loss of blood. In highly vascular intracranial processes the pressure is reduced at the start of operation.

(3) *Intracranial rise of pressure and cerebral edema.* Following administration of hexamethonium the intracranial pressure falls and peritumoral and general edema decreases. The operation can be carried out more rapidly, traumatization of the cerebral tissue is reduced, and the risk of postoperative cerebral edema is lessened.

CONTRAINDICATIONS

Intravenous administration of ganglion-blocking agents will obviously inhibit a number of the body's protective mechanisms. Our aim, therefore, has been to use such agents only in cases in which operations under controlled hypotension would be less distressing for the patient and less hazardous than operations without hypotensive agents.

If a patient with an arterial aneurysm has hypertension, this latter is not a contraindication, since the risk of rupture of the aneurysm and the consequences thereof during an operation without controlled hypotension will be greater than whatever damage may result from a blood pressure fall of short duration.

If a patient with a highly vascular intracranial tumor has arteriosclerosis and hypertension, he will assuredly run less risk at an operation under controlled hypotension induced by hexamethonium—since the bleeding will be easier to combat—than he would if bleeding resulted in shock and vasoconstriction. Hypotension with vasodilatation should give a better capillary circulation and, if the blood is well oxygenated, a more satisfactory oxidation of the tissues.

Grave cardiac and renal changes, pulmonary and extrapulmonary processes that considerably impair the oxygen uptake, and shock or impending shock are contraindications.

The drugs should accordingly be used to preclude bleeding, and not employed too late as a last resort when the hemorrhage has already served substantially to reduce the circulating blood volume.

COMPOSITION OF SERIES

The present series comprises 60 patients, 26 males and 34 females, operated on at the Department of Neurosurgery, Serafimerlasarettet, between January and November 1952.

Diagnosis	*Number of Cases*
Arterial aneurysm	28
Arteriovenous aneurysm	4
Meningioma	15
Neurinoma	5
Glioma	3
Intracranial metastases	2
Chordoma	1
Jacksonian epilepsy	1
Malignant hypertension (hypophysectomy)	1

The youngest patient was aged 5 and the oldest 63, the age incidence being as follows.

Age	*Number of Cases*
20 years or under	3
21–40 years	19
41–60 years	36
Over 60 years	2

Over one-half of the patients showed a good pre-operative condition; in one-third the general condition was affected by the disease itself or by other pre-operative complications; and 7 were in a very poor pre-operative condition, 5 being mentally confused and 2 unconscious.

Fifteen patients had hypertension. One had, if anything, a subnormal blood pressure in relation to the age.

Pre-operative electrocardiograms were taken in 48 of the 60 cases. In 27 cases the findings were normal; in 8 they showed very slight or slight aberrations; in a further 8 there were more pronounced, though still relatively mild pathologic changes; and in 4 the electrocardiograms were definitely pathologic, with low T waves in general, negative T waves, and ST-T depressions suggestive of coronary insufficiency and diffuse myocardial damage.

Thirteen of the patients showed hemoglobin values below 80 per cent, including 3 under 70 per cent.

Seven were operated on under local anesthesia and premedication alone.

Local anesthesia was induced in all cases—in 24 *without* supplementary exadrine.

The ganglion-blocking agents employed consisted of Hexathide (Allen & Hanbury) in 51 cases and Dytensin (Leo) in the others. The doses administered ranged from 10 to 200 mg.; namely, in 16 cases 20 mg. or less; in 26 cases 20–50 mg.; and in 18 cases over 50 mg.

In most (47) cases the head of the operating table was raised during some part of the hypotension period, though never to an extreme angle.

Initial effect of hexamethonium on the systolic pressure

12 cases	under 20 mm. Hg
6 cases	under 50 mm. Hg
19 cases	under 60 mm. Hg
11 cases	under 70 mm. Hg
9 cases	under 80 mm. Hg
3 cases	under 100 mm. Hg

Duration of hypotension under 70 mm. Hg

14 cases	1–15 min.
9 cases	16–30 min.
9 cases	31–45 min.
9 cases	46–60 min.
6 cases	1–2 hrs.
1 case	over 2 hrs.

Pulse during hypotension period

38 cases	unchanged
10 cases	bradycardia
8 cases	tachycardia

In 8 cases the blood pressure was raised solely by lowering the head of the table; in 40 cases fluid, Macrodex or blood transfusions were given too. In 12 cases, hypertensors—Oxedrin or L-noradrenaline—were administered as well.

Stabilization of the blood pressure postoperatively

3 cases	within 1 hr.
8 cases	1–2 hrs.
23 cases	2–4 hrs.
13 cases	4–6 hrs.
4 cases	6–8 hrs.
6 cases	over 8 hrs.
3 cases	not stabilized

Duration of operation or anesthesia

2 cases	under 2 hrs.
15 cases	2–3 hrs.
36 cases	3–4 hrs.
7 cases	over 4 hrs.

Complications During Operation. Apart from extreme falls in blood pressure no serious complications that could be attributed to the hypotension occurred during operation, except that the respiration was affected in 2 cases and a transient pulse arrhythmia was noted in 1, during the hypotension period.

Electrocardiograms were taken during the course of operation in 14 cases. Nine of them were consistent with those taken pre-operatively. In 1 case there was a very slight accentuation of the pathologic changes present in the pre-operative electrocardiogram, and in another case, which prior to operation had shown a normal electrocardiogram, the Q-T time became abnormal during hypotension. In 3 cases which before operation had shown pathologic electrocardiograms with low T waves and ST depressions, normalization occurred during the operation. A possible explanation of this is that the pre-operative electrocardiographic changes had resulted from psychogenic causes, with increased sympathetic tone which at operation was abolished by the ganglion-blocking agent.

Postoperative Course. Fifty-five patients were discharged after an average hospitalization of 15 days. Five patients died. In 1 of these the induced hypotension and subsequent treatment were contributory causes of death; the case is therefore presented in the following brief report:

Female, aged 57, with parasagittal meningioma, whose pre-operative condition was relatively good. Hypertension 160/100.

The operation was performed under insufflation anesthesia. Fifteen minutes after the operation had commenced, 10 mg. Hexathide were injected. The systolic blood pressure fell within a few minutes from 150 to a pulseless state. Noradrenaline was administered in drips, and soon afterwards the pulse was again palpable. The systolic pressure remained below 50 mm. Hg for about 15–20 minutes, below 70 mm. Hg for about 80 minutes, and below 80 mm. Hg for almost 2 hours. Blood, glucose and physiologic saline, as well as a total of *25 mg. noradrenaline* were administered

during the operation. In the next 36 hours at least *20 more mg. noradrenaline* were administered in drips.

Reeoperation was done in the evening of the same day. Pronounced cerebral edema was found.

Postoperatively there was peripheral edema and, in the leg in which a vein had been exposed for the intravenous drip, there gradually appeared a purple discoloration and the soft tissues necrosed in an area the size of the palm.

The patient died 14 days after operation, with rising N.P.N. and terminal bronchopneumonia. The autopsy findings included a groove, corresponding to the dural flap, in which local edema had evidently been present and had swelled the brain. The whole of the right hemisphere was edematous and in the operative field there was extensive malacia.

The other 4 deaths were caused by the disease.

Postoperative Complications in the Fifty-Five Patients Discharged. Four of the discharged patients underwent reoperation for *postoperative hematomas;* in 2 cases there were extradural and in 1 intracerebral hematomas. In 1 of these cases the hemorrhage was caused by slipping of the ligature, resulting in a subdural hematoma.

In no case were *pulmonary complications* observed. In 1, however, tracheotomy was done in order to facilitate respiration.

Postoperative shock occurred in no case.

Vomiting, either immediately or during the first 24 hours after operation, occurred in 23 cases. In 1 of them it continued during the next few days.

Postoperative ileus was not observed in any case.

The *postoperative urinary output* was satisfactory in all the cases and in no case was pathologic elevation of the N.P.N. level found.

Postoperative electrocardiograms were taken in 52 of these cases. Twenty-eight electrocardiograms, taken immediately after or the day after operation, either showed nothing abnormal or were consistent with the pre-operative ones. In 21 cases there were slight postoperative electrocardiographic changes, and in 3 more pronounced changes. However, all electrocardiograms were normalized prior to discharge.

EXPERIENCE

The hexamethonium compounds in intracranial operations were found to be effective agents for lowering the blood pressure, reducing bleeding and edema in the operative field, and, in operations on arterial aneurysms, lessening the risk of rupture.

It was feasible to use induced hypotension both at operations under local anesthesia alone and at operations under general anesthesia. We preferred, however, insufflation anesthesia as a routine method, so as to maintain the patient's airway and to ensure full oxygenation.

We were able to use local anesthesia *with* supplementary exadrine without any untoward effects on the hypotension or the postoperative course.

By restricted initial administration of the ganglion-blocking agent, it was possible to arrive at the effective dose for each individual patient.

Hypertensive patients reacted with greater falls of blood pressure in relation to the size of the dose.

Only in exceptional cases was the head of the table raised well above the horizontal plane; this in contrast to what the French authors Guiot and Damoiseau, among others, have reported at their operations. We found it unnecessary to have the patient in a semi-reclining position, which in our opinion is too hazardous.

The blood pressure should be allowed gradually to return to normal values. In our experience noradrenaline should not be employed as a hypertensor in intracranial operations. If lowering of the head and intravenous administration of fluid fail to rectify extreme hypotension, it seems physiologic, theoretically, to resort to noradrenaline so as to induce vasoconstriction. Noradrenaline has a pronounced vasoconstrictor action on all except the coronary vessels. Its action on the blood pressure is, however, in our experience, too difficult to control—especially after the use of ganglion-blocking agents. It must be borne in mind that its effect on the blood pressure in such cases will be further amplified since one of the mechanisms directed towards maintaining the pressure at a normal level is inhibited. In consequence we find blood pressure peaks and concomitant pronounced swelling of the brain. Attempts have been made to induce corresponding phenomena in experimental animals (cat and dog), though without any consistent results. Following repeated observations in the operating theaters, however, we have refrained from using noradrenaline in threatening situations. If, notwithstanding the controlled hypotension, serious bleeding has occurred and we have been unable to compensate this by intravenous administration, or to raise the blood pressure in the manner prescribed, we have resorted instead to intra-arterial transfusion of blood under positive pressure. In this way the impending irreversible shock has been rectified almost immediately and normal systolic pressure restored.

SYMPATHECTOMY OF THE UPPER EXTREMITY

EVALUATION OF SURGICAL METHODS*

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THE failure to obtain consistently good results in sympathectomy of the upper extremity and more particularly of the hand has been the cause for much speculation for over twenty years. A variety of factors, anatomical, physiological and pathological, have been highlighted from time to time and have resulted in changing modes of surgical operations. In view of accumulating evidence that some of the past reasoning regarding the anatomy and physiology of the sympathetic nervous system must be abandoned it is appropriate now to take stock of our surgical methods.

Three principles have long been mentioned as necessary in evaluating an operation on the sympathetic nervous system: (1) The operation must be anatomically complete, leaving no sympathetic nerves to supply the part to be denervated; (2) it must be extensive enough or performed in such a way that regeneration of sympathetic nerves to the part is obviated; and (3) it should be preganglionic, thus leaving intact the postganglionic supply to the part. A considerable bulk of the literature on the subject has to do with affirming the merits of one type of operation over another in meeting these proclaimed principles. While there is no fault to find in the principles, the interpretation of each of them has changed considerably. They will be considered in reverse order.

SENSITIVITY OF DENERVATED ARTERIOLES

Sensitization or supersensitivity of denervated tissue to circulating hormonal substances after sympathectomy was first described by Meltzer and Auer[20] and later supported by Cannon and co-workers,[8,9] who demonstrated that adrenin was the substance in question. Additional evidence of the overresponse of sympathectomized blood vessels to epinephrine was produced by experiments in man by Freeman, Smithwick and White[12] which led to the proposal that sensitization was one cause of failure of sympathetic operations for Raynaud's disease.[31]

From animal experimentation, Hampel[14] concluded that smooth muscle was less sensitive to chemical stimuli after preganglionic as opposed to postganglionic sympathectomy. The clinical evidence of the superiority of preganglionic sympathectomy in man seemed to exist in the fact that the results

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of the standard lumbar sympathectomy, which is a preganglionic operation, usually produced an effect in the lower extremity which was relatively better than that usually obtained in the upper extremity following the standard cervicothoracic or stellate ganglionectomy, which is a postganglionic operation.[34] In further support of this deduction Ascroft[4] showed in laboratory animals that by converting a preganglionic to a postganglionic sympathectomy in the lower extremity the reaction of blood vessels to adrenalin was increased.

Telford[32] in 1935 devised a technique for producing a preganglionic denervation of the upper extremity in man. His operation was performed through a supraclavicular incision with division of the clavicular head of the sternocleidomastoid and the anterior scalenus muscles. The rami of the 2nd thoracic ganglion were severed and the paravertebral chain was divided below the ganglion. The rostral end of the divided chain was then anchored in the cervical muscles to prevent regeneration of fibers that might bridge the gap between the ends of the divided chain. Later he extended the operation to include the 3rd thoracic ganglion.

Smithwick[29] (1936) independently developed a preganglionic sympathectomy comparable to Telford's but performed through a posterior rib resection. Smithwick's operation entailed division of the rami of the 2nd and 3rd thoracic ganglia and division of the chain below the 3rd ganglion. The rostral end of the divided chain was swung out of the thoracic cavity and sutured into the paravertebral muscles; the distal end was ligated. In addition, segments of the 2nd and 3rd intercostal nerves and their intradural roots were resected to prevent regeneration from this source. As a later addition to the operation, capping of the cut ends of the chain was proposed further to minimize the possibility of regeneration. A still later addition has extended the operation to include the 4th thoracic ganglion and intercostal nerve.

Thus the principle of the advantage of the preganglionic over the postganglionic sympathectomy and the development of Telford's and Smithwick's operations more or less guided thinking and surgical methods for some years thereafter, and corroboration was forthcoming from many sources, as Learmonth,[17] de Takáts and associates[25] and Atlas,[5] to name but a few.

But there were at least two studies made during this period that questioned the importance of a difference between preganglionic and postganglionic sympathectomy in man. Simmons and Sheehan,[27] in exploring the cause of relapse following sympathectomy of the hand, reviewed 38 ganglionectomies and 29 preganglionic sections in Telford's clinic. They did not find any significant difference in the number of relapses following the two types of operation and concluded that regeneration of vasoconstrictor fibers is the probable cause of relapse after either type of sympathectomy.

The other study was by Fatherree, Adson and Allen[10] on the vasoconstrictor action of epinephrine on the digital arterioles of man before and

after sympathectomy. They found that in testing for sensitivity with adrenalin the methods employed were not wholly reliable because of marked differences in responses under apparently identical circumstances. But within the limits of their tests they demonstrated that preganglionic and postganglionic section of the sympathetics to the hands increase the sensitivity of the digital arterioles to intravenous injection of epinephrine in about the same degree. Their disbelief that sensitivity of the denervated arterioles to epinephrine has anything to do with failure to cure all patients with Raynaud's disease of the hands was the reason for Adson's[2] continuing his preference for cervicothoracic ganglionectomy.

Several reports in recent years[6,11,15] have emphasized that recurrence of vasospasm almost never occurs without the presence of vasomotor nerve supply. It remained for Simeone and his associates[26] to terminate the preoccupation that has existed too long with this problem. They pointed out that denervations of the upper limb in man are not completely postganglionic even when the stellate ganglion is removed, because of postganglionic cell bodies in the middle cervical ganglia and possibly elsewhere. On the other hand, the preganglionic operations probably include a variable portion of postganglionic fibers among those interrupted. They concluded that any increased sensitivity of the blood vessels that occurs in the early period after sympathectomy is not important, that sensitivity diminishes as regeneration of vasomotor fibers occurs and that return of Raynaud's symptoms is the result of this regeneration.

It can be assumed, then, that if there exists any difference in the degree of sensitivity of arterioles to circulating epinephrine after a preganglionic and a postganglionic operation the difference is unimportant, at least in man, and the sensitivity after either operation has nothing to do with clinical relapses. Relapses are believed to be the result of other factors that perpetuate or restore activity of the sympathetic nerves.

REGENERATION OF SYMPATHETIC NERVES

The powers of regeneration of sympathetic nerves in experimental studies of certain laboratory animals are well known, although a marked species difference has been noted.[7] In some animals there is a remarkable property of regeneration of the cervical sympathetic chain even when long sections are removed and muscle is interposed between the cut ends.[16,18] The short preganglionic rami of the stellate ganglion regenerate within several weeks in cats.[33]

White and Smithwick[35] reported that with secondary operations in man and removal of paravertebral scar tissue after preganglionic sympathectomy, sympathetic activity which had recurred was again abolished and led to the conclusion that regeneration is an important cause of failure in attempts to sympathectomize the upper limb. But at the New York Hospital secondary operations of this kind were abandoned because the results reported by White and Smithwick could not be duplicated.

There has been a general interest in possible examples of regeneration of sympathetic nerves in man elsewhere in the body since by correlation some light might be thrown on the question. Smithwick[35] reported having witnessed restitution of previously resected short lengths of major splanchnic nerves. This is perhaps what might be expected yet in my experience in 3 cases, 2 to 5 years after supradiaphragmatic division of the splanchnics and lower thoracic sympathetic trunk, examination failed to show gross or microscopic nerve connections; similar findings were reported by Adson.[1] The resection of 3 cm. of the mid portion of the cervical sympathetic trunk for removal of a benign tumor has not resulted in regeneration after 6 years even though pains were taken at the time to prevent retraction or misplacement of the cut ends.

My experiences do not prove that regeneration of preganglionic fibers does not occur in man, only that it does not always occur even under favorable circumstances. In particular, it seems likely that in the small confines of the upper thoracic region preganglionic fibers traversing but a short distance from their cells of origin in the cord to the intact stellate ganglion might readily regenerate in man. The same should be true for regeneration of fibers from the distal end of the divided upper thoracic trunk, yet more evidence that regeneration actually occurs would be welcome.

After cervicothoracic ganglionectomy which removes the postganglionic cell bodies it is believed to be physiologically impossible that preganglionic fibers could join with postganglionic neurons or grow down the arm and take over the function of innervating smooth muscle. There is a possibility that the middle cervical ganglion, if left intact, could serve for a postganglionic connection for regenerating preganglionic nerves, and this ganglion should probably be removed in performing a postganglionic sympathectomy. However, in one of my patients in whom ganglionectomy was performed from the mid cervical to the 3rd thoracic ganglia on one side, and from the stellate to the 3rd thoracic on the other, there was the same degree of residual sympathetic activity in the two hands one year after operation.

Livingston[19] suggested that a considerable number of postganglionic cells related to the arm lie in the ganglia below the 2nd thoracic and that regeneration of postganglionic nerves from these might occur. The possibility exists but in our (Ray and Console[23]) 30 patients with total sympathectomy which includes excision of the paravertebral chains from the stellate to the 3rd lumbar ganglia inclusive, there remained unquestioned sympathetic activity in the hands.

In brief, the great variety of surgical methods employed in sympathectomy of the upper limb and designed to prevent regeneration of sympathetic nerves has not been effective in producing complete and permanent sympathectomy. This fact obtains in both the preganglionic and postganglionic types of operation. There is no basis then for concluding that regeneration is the cause of incomplete sympathectomy to the exclusion of other possible causes.

A paradox seemingly exists in the lower extremity where the standard operation of resection of the lumbar paravertebral chain is followed by complete and permanent sympathectomy, at least from the knee down and in additional areas on the posterior thigh. In studies by Ray and Console[22] no exceptions were found in a group of patients examined several years after operation. While occasional exceptions may exist because of anomalous innervation the probable explanation for such exceptions is that the operation was technically at fault. The lumbar sympathectomy is principally if not wholly preganglionic since there are numerous postganglionic cells in the lowest lumbar and the sacral paravertebral ganglia. If regeneration were a factor in the return of function in the upper limb it would seem that regeneration might also occur often from the divided preganglionic lumbar rami or from the end of the resected lumbar chain and restore sympathetic innervation in the leg and foot. Yet, this does not occur.

The inference is clear that the standard operation of resection of the lumbar paravertebral chain interrupts all the sympathetic nerve supply to the leg and foot and regeneration does not occur. Whereas, in the upper limb any combination or refinement of preganglionic or postganglionic operation yet devised is inadequate.

COMPLETENESS OF SYMPATHETIC DENERVATION

The foregoing discussion and deductions lead logically to consideration of the question of completeness of the interruption of all anatomical sympathetic connections in sympathectomizing an extremity. Investigation was made of the effect of sympathectomy in the lower extremities in 151 patients having 291 operations.[22] The convenient method was employed of measuring sympathetic activity by determining the skin resistance, though supplementary and confirmatory observations were made on surface temperature and of changes in color in the skin. The following were among the conclusions reached:

(1) After excision of the lumbar paravertebral ganglionated chain, regardless of the upward extent of the excision, sympathetic innervation is still present in a part of the lower limb. (This was referred to as the result of "residual innervation.")

(2) The nerves providing residual innervation do not traverse any part of the paravertebral chain.

(3) Sympathetic activity is present in all patients in the anterior thigh (i.e. in dermatomes T12 to L3) after lumbar or even after total paravertebral sympathectomy and may be completely interrupted by the addition of sympathetic blocking agents, spinal anesthesia, procaine block or division of lumbar nerves, or by anterior rhizotomy of T12, L1 and L2. The rhizotomy performed in conjunction with paravertebral sympathectomy results in complete and lasting sympathectomy of the thigh.

(4) After lumbar paravertebral sympathectomy the remaining or "residual" nerves which provide innervation to the anterior thigh manifest their

activity only after a process of readjustment or augmentation takes place.

(5) An incomplete denervation may therefore mimic complete denervation for a period up to 3 months.

The degree of augmentation in sympathetic activity that takes place in undenervated areas after other parts of the body have been sympathectomized is impressive. Though its occurrence is universally recognized its importance is not commonly realized. Evidence of the effect is often seen in the increased vascular tone that develops in one limb after the opposite has been sympathectomized or, more dramatically, in the development of Raynaud's changes in the hands after thoracolumbar sympathectomy.

In patients having total bilateral paravertebral sympathectomy from the stellate to the 3rd lumbar ganglia the retained activity in the anterior

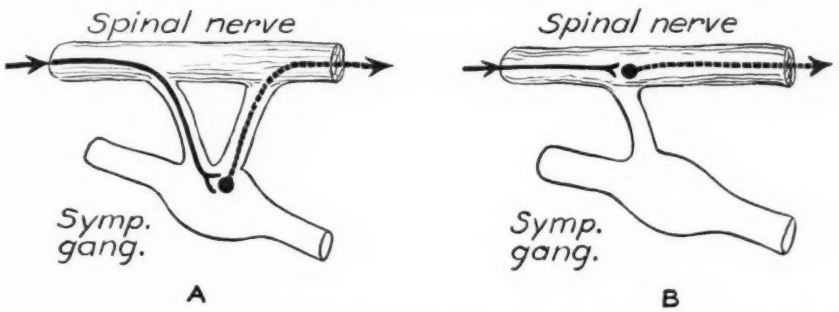

FIG. 1. Showing variable arrangement of synapse between preganglionic and postganglionic neurons. (A) Usual arrangement, in which the synapse takes place in the paravertebral ganglionated chain and pre- and postganglionic neurons traverse rami communicantes. (B) Arrangement described by Wrete[36] and by Skoog,[28] in which the synapse occurs outside the paravertebral chain through "intermediate ganglia."

thighs becomes excessive as evidenced by the presence sometimes of drenching sweat and the appearance in some of livid blotching and coldness of the skin in these areas. Also, and without exception, patients with total paravertebral sympathectomy show cold hands with patchy areas of sweating and a tendency to develop color changes. Other regions, including the face and scattered areas over the trunk, show similar effects, the excessive activity which appears early in some parts becoming less as augmentation in remaining innervated areas develops.

The significance of the investigations of Wrete,[36] of Skoog[28] and more recently of Alexander, *et al.*,[3] which provide an anatomical basis for explaining the incompleteness of paravertebral sympathectomy, has been only lately appreciated. Wrete demonstrated in man the existence of "intermediate" ganglia in the rami adjacent to the 1st and 2nd lumbar nerves. Skoog, working in Wrete's laboratory, found similar ganglia attached to cervical and upper thoracic spinal nerves in man. The ganglia in many instances were virtually incorporated in the spinal nerves. Skoog believed

that the fibers connecting these intermediate ganglia constituted a preganglionic and postganglionic pathway confined wholly to the spinal nerve and unconnected with the paravertebral chain. The intermediate ganglia were most commonly found by Skoog in the 8th cervical and upper two thoracic spinal nerves, but were also found accompanying higher cervical nerves. He suggested on the basis of his anatomical investigation that the early relapse after sympathectomy of the upper limb would thus be explained by the fact that important pathways were not severed by the operation. He considered the immediate postoperative effect of apparent complete loss of sympathetic activity to be comparable to "local shock" brought about by severing several neurons but soon compensated for by the few nerves that remained intact.

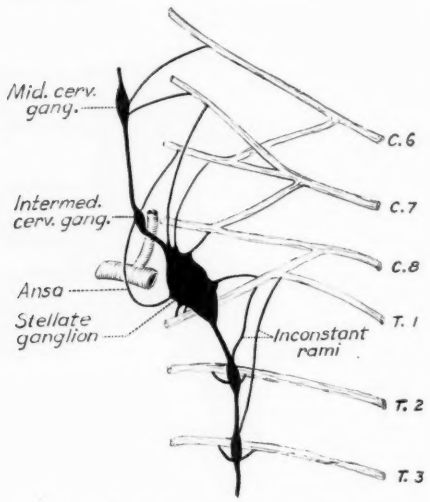

FIG. 2. Semischematic drawing of the cervicothoracic ganglia and chain to be removed in the operation recommended for sympathectomy of the upper limb.

In the development of his operation for sympathectomizing the upper limb Smithwick recognized the probable advantage of resecting the 2nd, 3rd and 4th intercostal nerves and their roots but preserved the 1st thoracic, maintaining that a sympathetic outflow to the upper limb is absent or insignificant at this level and cannot be sacrificed without undesirable effects.[30] But in view of the existing evidence, the importance of the 1st thoracic and very probably of some cervical spinal nerve pathways can no longer be ignored.

At the New York Hospital we[24] have in 3 patients combined cervicothoracic sympathectomy, from the inferior cervical to the 3rd thoracic ganglia, with intradural rhizotomy of the first three thoracic anterior roots. In 2 of these patients there was no significant motor deficit in the extremity while in the third, probably because of a "post-fixed plexus," there was some permanent motor loss in the hand. Postoperative tests indicate that even with this operation there is evidence of some sympathetic activity in the hand and the assumption is that there remains an innervation traversing cervical spinal nerves.

We[24] attempted to go further with the investigation and in several patients recording of skin resistance and plethysmographic measurements were taken from the hand on stimulation of the lower cervical and first thoracic anterior roots at the operating table. Because of the difficulties imposed by

the strong muscle contractions in the hands and fingers resulting from stimulation of the anterior roots the determinations of sympathetic responses have not been conclusive. However, there are definite suggestions in the records of positive sympathetic response from stimulation of the 7th and 8th cervical and 1st thoracic roots.

DISCUSSION AND CONCLUSION

The accumulating bulk of evidence points strongly to the existence of a sympathetic supply to the upper limb so extensive that no practical operation, either preganglionic or postganglionic alone or in combination, can be relied on to effect an absolutely complete denervation. Complete denervation theoretically could be assured only by sectioning motor roots important to muscular function of the limb. Even so, the various preganglionic or postganglionic operations currently employed for sympathectomizing the upper limb often produce good clinical results though they fall short of being perfect.

There is no need to abandon sympathectomy for appropriate cases of vascular disorders of the upper extremity but there is need for clear thinking on the matter of the most effective and practical way to perform the operation within the limits of the possibilities.

It appears that the 1st thoracic complex of root, nerve and ganglion has a part in the innervation of the extremity. A premium can no longer be put on preservation of the 1st thoracic ganglion in order to avoid a miosis and ptosis of the upper eyelid. A symmetrical bilateral Horner's syndrome is not disfiguring and is not objectional to most patients. If in an occasional patient only one upper limb is to be sympathectomized and it is important to have a symmetrical appearance of the eyes a superior cervical ganglionectomy can be performed on the opposite side without adding materially to the operative procedure.

If the evidence is accepted that there is little, if any, significant difference in the sensitivity to circulating epinephrine of the arterioles following preganglionic and postganglionic sympathectomy in man there is no need to insist on the former.

Also, if there exists even the theoretical possibility of regeneration after preganglionic sympathectomy it can be obviated by a paravertebral ganglionectomy that is extensive enough to remove the cells of the postganglionic neurons.

This line of reasoning leads to the inevitable conclusion that resection of the cervicothoracic ganglionated chain will result in:

(1) No significant increase in sensitivity of the arterioles of the upper limb.

(2) Prevention of regeneration through removal of the cells of the postganglionic neurons.

(3) As complete a sympathetic denervation of the upper limb as can be accomplished, short of paralyzing the limb.

Since there are probably postganglionic cells sometimes in ganglia in the cervical trunk above the stellate ganglion, it may be desirable to carry the excision upward to include the middle cervical ganglion in patients in whom this ganglion is present. The lower extent of the excision should include at least the 3rd thoracic ganglion.

A resection of this extent can best be performed through an anterior supraclavicular incision similar to that described by Gask and Ross.[13] A resection of less extent that would spare ganglia above the stellate ganglion could also be performed through a posterior approach, such as is employed for the preganglionic type of operation. This approach is a more mutilating operation and results in a more stormy convalescence. If intercostal nerves are sacrificed in the latter operation the resulting dysesthesia may overshadow any benefit of the sympathectomy in the patient's mind.

But it is never profitable to attempt to standardize ways of performing any operation. The purpose of this discussion is merely to establish that the postganglionic operation of resection of the cervicothoracic chain from the middle cervical to the 3rd thoracic ganglia is as good as any devised preganglionic operation and probably better. It is time to cease insisting that sensitization and regeneration constitute the chimera that destroys the good effects of our operations.

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SURGICAL TREATMENT OF ANEURYSMS OF THE ANTERIOR COMMUNICATING ARTERY

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A STUDY of the literature shows that there has been increasing interest in the surgical treatment of intracranial aneurysms, largely because of the extremely poor ultimate prognosis of conservative management in cases of subarachnoid hemorrhage from this source. With accumulating experience, the surgical mortality is improving and more authors are coming to the opinion that surgical attack of one sort or another is the method of choice in the approach to this problem. Interest has therefore been aroused

TABLE 1

24 surgically treated aneurysms of the anterior communicating artery

Procedure	No. of Cases	Op. Deaths	Subsequent Death from Subarachnoid Hemorrhage
Ligation of carotid artery	2	0	0
Exploration only	2	0	1 (1½ yrs. later)
Muscle or gelfoam packed around aneurysm	4	0	1 (gelfoam case, 4 wks. later)
Trapping	1	1	
Ligature of aneurysmal neck with thread or clip (in 1 case aneurysm then excised)	15	1	
Total	24	2	2

as to the types of procedure effective in these cases and the specific indications for each in an effort to combine maximum result with minimum of hazard.

Because of their unique location on the major anterior channel for collateral circulation in the circle of Willis, we feel that aneurysms of the anterior communicating artery represent a special problem in the surgical treatment of intracranial aneurysms. In spite of their infrequent separation as a distinct category in the literature, they are not properly comparable to other intracranial aneurysms either in assessment for surgery or in choice of surgical procedure to be employed.

The present material comprises 26 cases of aneurysms originating from

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the anterior communicating artery and represents the entire unselected material of this classification from this clinic up to April 1952. Of these patients, 24 were subjected to surgery of one sort or another (Table 1). It is the purpose of this paper to review the problem of the surgical treatment of aneurysms located on the anterior communicating artery, and in light of our experiences and those of others, to assess which approach may be the most rational and at the same time most rewarding.

INCIDENCE

The incidence of intracranial aneurysms in the general population has been estimated up to about 1 per cent;[19,22] and in almost all recorded series[1,3,4,11,13,23,28] aneurysms of the anterior communicating artery represent somewhere between 20–25 per cent of them. The 26 aneurysms of the anterior communicating artery here recorded represent slightly over 20 per cent of all aneurysms recorded at the Serafimer clinic since 1932.

The importance of recognizing any specific problems presented by such a group is thus clear.

ETIOLOGY

Views as to the etiology of these "congenital" aneurysms have varied. Forbus[12] felt that their development was caused by the absence of media at the bifurcation of the arteries which led to a weak point that gradually gave way under the constant pulsating pressure of the blood within at these points. In support of his theory was the high incidence of aneurysms at the points of bifurcation on the circle. It did not, however, explain those aneurysms that occurred not at all infrequently at a point along the course of a vessel and well away from its bifurcation, most notably those on the proximal part of the carotid and, more germane to our present discussion, at the midpoint of the anterior communicating artery.

On the basis of some very convincing investigations into the embryology of the circle of Willis, Dorcas Padget[4] pointed out that the development of aneurysms could more logically be traced to weak points occurring at the sites of incomplete resolution of previously existing embryonic vessels. From the anterior communicating artery in the embryo, there arises a smaller median anterior cerebral artery. This normally disappears, but De Vriese[5] found it to be present in a large percentage of human embryos and called it the "median artery of the corpus callosum." In adult man it may anomalously persist as a single anterior cerebral artery with usually a relative disappearance of the two normally well developed anterior cerebral arteries. In some vertebrates, this median artery normally persists. It is the incomplete resolution of this artery that may be the point of origin of the aneurysms of the anterior communicating artery. In any event, Padget's investigations led Dandy to the belief that this embryological explanation was the more logical, and Bassett[2] has recently re-emphasized the point of etiology as most likely "unresolved vestiges of a primitively normal circulatory system" as opposed to the theory of Forbus. The present authors

are inclined also to this view. Of our 26 cases, in only 9 was there any question that the aneurysm arose from the communicans trunk well away from the point of junction of the communicans with either anterior cerebral.

SYMPTOMATOLOGY

It is beyond the scope of this paper to discuss symptomatology in detail and our series is hardly large enough to warrant conclusions. There are, however, one or two points that have struck us as characteristic and they are worth brief mention. As with other intracranial aneurysms, the usual story is of no symptomatology whatsoever until the occurrence of a sudden subarachnoid bleeding with its familiar and typical signs. Of our 26 patients 24 had subarachnoid hemorrhage and the number of episodes varied from 1 to 3. Paralytic signs were surprisingly frequent, occurring in 10; 4 of these patients had partial paralysis (arm and/or face or leg only). We were unable to correlate the side of paralysis with the projection of the aneurysm and were surprised to find that only 3 of the 10 had intracerebral hematomas that could explain the paralysis. Richardson and Hyland[28] had noted focal signs in 62 of their 118 cases which in most instances were caused by concomitant intracerebral hematoma. The presence of a hematoma had also been felt by us to be the most likely etiology of a hemiparesis. A more interesting and suggestive correlation is that of actual narrowing of a vessel or nonfilling of a specific vessel at arteriography. Ecker and Riemenschneider[7] hypothesized that this narrowing of a vessel in cases of aneurysms

FIG. 1. Aneurysm of the anterior communicating artery. Note spasm of the carotid artery, middle cerebral artery and anterior cerebral artery (arrows).

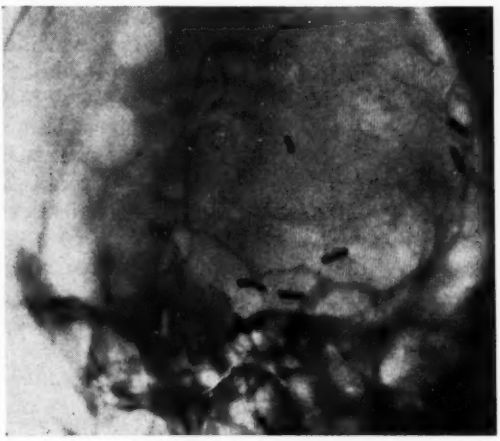

FIG. 2. The same case as in Fig. 1 after operation. Note clip on the neck of the aneurysm and disappearance of vasospasm.

is in all probability caused by arterial spasm, and postoperative angiograms in our series have demonstrated disappearance of this narrowing, tending to confirm their opinion (Figs. 1 and 2). The direct operative visualization of the anterior cerebral artery as a thin white thread in some cases in this series has suggested severe spasm or congenital defect as a possible explanation.

Of the 10 patients exhibiting paralytic signs, 7 had either arterial spasm or nonfilling of a vessel and in each instance the vessel involved was the one that anatomically would give rise to the symptomatology observed (Table 2). Thus, the patients with involvement of face and arm or leg alone showed spasm of the anterior cerebral artery or nonfilling on the opposite side (see also Ethelberg[10]) and the patients with complete hemiparesis showed spasm of the carotid on the opposite side. Of the 3 patients who had hemiparesis and no spasm or nonfilling, 1 had an intracerebral clot of the opposite frontal lobe which could have accounted for the symptomatology. The etiology of the other 2 is obscure.

We are all aware of the limitations of arteriographic evidence of spasm of a vessel, especially when 35 per cent umbradil (diodrast) rather than thorotrast is used and are also sure that absence of filling of a vessel (especially the anterior cerebral) on one examination cannot be used as an indication of its being in spasm, congenitally absent or thrombosed. However, the correlation of direct visualization of arterial spasm and/or nonfilling and the signs resulting indicate to us that this is more than coincidence. We would therefore suggest that the focal paralytic signs seen in our cases were caused by vasospasm secondary to the existence of the aneurysm, the spasm possibly being a protective mechanism after bleeding. It will be of

interest to observe if future larger series can confirm this hypothesis.

As may be seen in Table 2, there were 6 cases in which there was arterial spasm of varying degree without clinical hemiparesis, but we would interpret this as being related to the duration and degree of spasm necessary to cause functional damage to cerebral substance. Also, in 5 of the 8 cases of nonfilling there were no clinical signs. It is obvious that there are some difficulties in correctly interpreting the significance of spasm or nonfilling of a vessel. For the reasons outlined above, however, we feel that there is a cor-

TABLE 2

Occurrence of vasospasm or arteriographic nonfilling of vessels and clinical paralysis in cases of aneurysm of the anterior communicating artery

Case No.	True Vasospasm	Nonfilling	Paralytic Sign
2	Rt. ant. cerebral	—	—
3	—	Rt. ant. cerebral	—
4	—	—	Slight lt. hemiparesis
5	Lt. carotid	—	Rt. hemiparesis
6	Rt. carotid	—	Lt. hemiparesis
7	Rt. carotid	Rt. ant. cerebral	—
9	—	Rt. ant. cerebral	—
10	Rt. carotid and middle cerebral	—	Lt. hemiparesis (temporary)
11	—	Lt. ant. cerebral	—
12	Rt. ant. cerebral	—	Lt. leg
13	—	Rt. ant. cerebral	—
14	—	Rt. ant. cerebral	Lt. arm and face
15	—	Rt. ant. cerebral	Lt. arm
16	Lt. ant. cerebral	—	—
17	—	Rt. ant. cerebral	Lt. arm
19	—	—	Rt. hemiparesis
20	—	—	Slight rt. hemiparesis and sl. aphasia
22	Rt. ant. cerebral	—	—
23	Rt. ant. cerebral	—	—
24	Lt. ant. cerebral	—	—
Total	10	8	10

relation of significance here, although actual proof of this must await further evidence.

We found absolutely no correlation between the site of vasospasm and the projection of the aneurysmal sac—that is to say an aneurysm projecting to the right was no more likely to produce spasm of the right anterior cerebral than one projecting to the left.

The sudden onset of blindness in one or the other eye was rather prominent as a finding, occurring in 7 (25.9 per cent) of our cases. This type of complaint seems rather characteristic for aneurysms in this location and we interpret the mechanism as one of sudden expansion of the aneurysmal sac with encroachment on the optic nerve.

ARTERIOGRAPHY

The necessity for complete bilateral carotid angiography in all of these cases cannot be overemphasized. This has been stressed also by List and Hodges,[20] Poppen,[27] and Löfstedt.[21] That angiography is not without its dangers is, of course, well accepted and the complications of the procedure have been much discussed, most recently by Dunsmore, Scoville and Whitcomb.[6] Two of our patients were worse after angiography. If, however, one intends to practice surgery in these cases, it is a rather small risk to take compared to the vital information obtained. Firstly, the co-existence of multiple aneurysms is well known. Dandy[4] found multiplicity in 15 per cent of his 108 patients, although Ask-Upmark and Ingvar[1] have rightly stated that there is "usually" only one. We are aware that angiography does not always demonstrate the aneurysm or all of the aneurysms. However, the angiographic evidence of multiplicity will be a deciding factor in which procedure to employ or if any at all should be attempted. None of our cases of anterior communicating artery aneurysm showed multiplicity.

It is now generally conceded that angiography early after an episode of hemorrhage does not essentially increase the morbidity, and the shortness of the interval from the acute episode should not influence performance of this examination. Our 2 patients who were worse after angiography had it performed 1½ weeks and 8 weeks respectively after the last episode of bleeding.

Secondly, it is imperative to establish the exact state of the collateral circulation between the hemispheres and to exclude anomalies of the circle of Willis before deciding to attack the aneurysm. This is especially important if carotid surgery or trapping is contemplated and also aids in an emergency decision should uncontrollable hemorrhage from rupture at operation occur. Padget[4] noted that in individuals with aneurysm, variations in the circle of Willis were about 2 times as frequent as in those without aneurysm. She felt that there was a high percentage of absent arteries in these cases. Jefferson[18] believes that the circle of Willis is incomplete in about 8 per cent of normals. In Falconer's[11] series, 5 out of 11 patients with aneurysm in this location had arteriographic evidence of anomalies of the circle of Willis "such that distal to the communicating artery, both anterior cerebrals were supplied by one carotid artery only, suggesting a deficiency in the circle of Willis between the anterior communicating artery and the internal carotid artery of the opposite side." One of these patients died with bilateral frontal lobe infarction after carotid ligation.

That the aneurysm itself may not fill from both sides appears to be the usual occurrence (Fig. 3). Of 20 cases in which bilateral arteriography was done, there were only 2 in which the aneurysm filled from both sides, the marked predominance of unilateral filling being from the left carotid (12 out of 18). The significance of this last observation is not clear, but is rather striking.

Thirdly, it is by angiography alone that the size, configuration, extension,

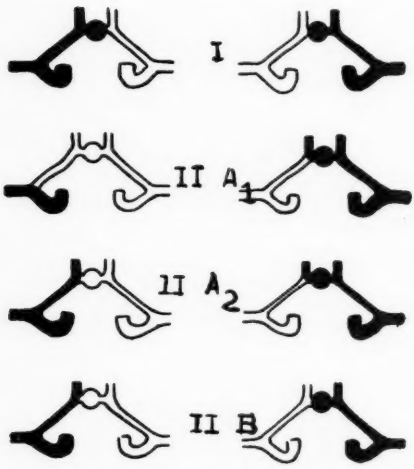

FIG. 3. Patterns of circulation through the anterior part of the circle of Willis in 20 cases of bilateral angiography.

I. Aneurysm filled from both sides; 2 cases.
II. Aneurysm filled from one side only; 18 cases.
A. Aneurysm and both pericallosal arteries filled from one side; 15 cases.
1. Proximal part of the anterior cerebral artery not filled at all from the opposite side; 8 cases.
2. The whole anterior cerebral artery filled from the opposite side; 7 cases.
B. Aneurysm and homolateral pericallosal artery filled from one side. Proximal and distal part of anterior cerebral artery but not aneurysm filled from the opposite side; 3 cases.

and position of the aneurysm itself can be determined—information of great value in the planning of a surgical approach. Clinical signs alone may be misleading as to location of the aneurysm as shown in our cases of hemiparesis. Walsh and Love[32] have noted ocular nerve palsy on the side opposite to an aneurysm and Jefferson[17] noted unilateral blindness on the contralateral side. Elvidge and Feindel[9] have mentioned the possibility of EEG helping in the lateralization, but have also pointed out that clinical and pneumographic evidence may indicate the wrong side because of a hematoma, which is not necessarily on the same side as the lesion producing it. This ability of midline rupture to go to either side is exemplified when we consider that the anterior cerebral arteries may lie one above the other as they pass upwards between the hemispheres, and hence either may rupture into the brain substance of either side.

Of the 19 cases of this series in which arteriography disclosed a definite projection of the sac, the projection was downwards and forwards in most instances, as in Falconer's[11] cases. We were unable, however, to correlate projection with symptomatology. The aneurysm was demonstrated in all 26 cases by angiography and was clearly stated to be anterior communicating in location by the roentgenologist prior to operation.

MANAGEMENT OF CASES

Results of Conservative Management. The prognosis of subarachnoid hemorrhage has been studied by several investigators. Ask-Upmark and Ingvar,[1] in a study of 138 patients, found that about 32 per cent died in their first attack and about 32 per cent had recurrence of hemorrhage, the mortality being higher at the second bleeding. They have also made what we consider a most important observation as to time of death, i.e., that death

from one subarachnoid hemorrhage may be in the first 24 hours or as late as the 25th day, the average length of time being the 9th day after the episode. This is a factor worth considering in evaluating procrastination in active therapy, once decided upon. They concluded from their own experience and a review of the literature that a patient with subarachnoid hemorrhage has only 1 chance in 5 of a good recovery. He has 1 chance in 5 of being permanently crippled and 3 in 5 of dying sooner or later from his disease.

This gloomy outlook of high mortality, frequent recurrence and increasing mortality with recurrence has been confirmed by others.[4,13,24,33] Hyland[16] reported in 1950 that of 191 patients with subarachnoid hemorrhage 100 died within 6 months of the onset of the attack. Falconer[11] has concluded from the literature that the mortality of conservative treatment of spontaneous subarachnoid hemorrhage is 50–60 per cent. He tabulated the figures of Taylor and Whitfield, Hamby, Hyland, Magee, and his own series to support this, and also showed the high incidence of recurrent attacks in patients *while under observation* (46 per cent of his and 51 per cent of Magee's patients). Again, the time factor seems to us to speak against the advisability of procrastination.

It has been the hope of conservative management that "healing" would take place at bed rest with abstinence from straining, etc. or that the aneurysm itself would thrombose. The above figures would seem to belie this hope. Dandy[4] noted that 2 of his series and several from the literature had *almost* filled with thrombus, but eventually ruptured from the small remaining lumen. He concluded that not more than 15–20 per cent of hemorrhages are cured permanently by thrombosis.

Results of Surgical Management. Although the results of surgical treatment of aneurysms are not yet brilliant, they are with experience constantly improving and, as compared with the results of conservative management, they are encouraging. We do not believe that a truly precise comparison between a series of patients with subarachnoid hemorrhage treated surgically and those handled by conservative therapy is possible at the present time. Falconer[11] has also pointed out the difficulties in obtaining series that are reliable for such a comparison but is of the opinion that in his series of 50 surgically treated bleeding aneurysms the mortality would have been much higher than 18 per cent had they been conservatively treated.

From different statistics on the prognosis of subarachnoid hemorrhage it is quite obvious that the time factor is of utmost importance. Actually, we are faced with two problems. The first is how to treat these patients in the acute stage of hemorrhage. The second problem is what should be done in the free interval, i.e. how to prevent recurrent bleeding. The acute stage covers about the first 3 weeks and corresponds to the period of vascular spasm.[7] It is the period of highest mortality.[1,28] In any analysis that deals with this question, it is therefore necessary to consider the interval between the bleeding and the operation.

Any surgeon is naturally disinclined to operate on a critically ill comatose patient and wonders if it is not wiser to wait until the general condition improves before subjecting him to the additional stress of a major procedure. On the other hand, the high mortality in the first 24–48 hours of all cases as well as the frequent recurrence with death even in patients doing well under observation leads one to a consideration of whether these patients should not be operated upon as soon as possible after their episode of hemorrhage. The optimal time factor for operation has recently been considered in detail by one of us.[26] As previously mentioned, early clinical symptoms may perhaps result from spasm of a vessel. We are convinced that in many cases the vasospasm is a factor in the coma following subarachnoid hemorrhage.

SURGICAL PROCEDURES

Carotid ligation does not at this stage of experience seem to us a very rational procedure for aneurysms of the anterior communicating artery, in spite of what its effect may be on aneurysms of other locations. Ligation of one carotid can hardly be expected to reduce the blood flow more than temporarily in the communicating artery which under normal conditions potentially receives its flow from both sides. In the event that this were not so because of poor collateral circulation, ligation of one carotid would carry extreme hazard to the nutrition of the cerebral hemisphere on that side. However, Krayenbühl[19] reported recovery from a bitemporal hemianopsia after carotid ligation in a patient with anterior communicating artery aneurysm, and Walsh and Love[32] ligated the right internal carotid in a patient who had had 2 episodes of bleeding from an aneurysm at the junction of the right anterior cerebral and the communicans with good results 3 months later.

On the other hand, Falconer[11] tried carotid ligation alone in 3 patients with anterior communicating artery aneurysm and 2 died. One had a vascular anomaly of the circle of Willis and ligation resulted in infarcts of both frontal lobes. In the other the choice of the side of ligation was determined by the presence of an intracerebral hematoma on that side, which actually had arisen from the opposite anterior cerebral. Moreover, he admitted that occlusion of one carotid can only temporarily decrease the pressure in the communicating artery, especially if the aneurysm can be filled from either side, and concluded that a direct intracranial approach was the method of choice for aneurysms in this location. The attractions of carotid surgery have been that it is easy to do, there is less operative risk, and there may perhaps be benefit. The use of carotid surgery alone, however, precludes the evacuation of intracerebral hematoma, which we feel of importance for the most favorable outcome in any case. Four of our 22 intracranially explored patients had intracerebral clots and 3 of these 4 were the most critically ill of our series. The 4th was disoriented prior to evacuation of the clot. Two clots were frontal, 1 was callosal, and 1 hypothalamic.

Very few aneurysms of this location operated upon by direct surgical

attack have been reported. Dandy[4] had only 4 in his group of anterior communicating and anterior cerebral artery aneurysms. Of Falconer's 11 cases a direct attack was undertaken in 8. Single cases of successful operation have been reported by Tönnis,[31] Russel,[29] and Elvidge and Feindel.[9]

In discussing the surgical procedure in these cases it should be mentioned that since August 1950, when a more active attitude towards these lesions was instituted in this clinic, in practically all cases induced hypotension by spinal anesthesia or by hexamethonium has been used during the operation. We are of the opinion that this has been of extreme value.

Unless a left-sided hematoma is suspected, we have preferred to use a high right frontal flap. As has been pointed out by Falconer,[11] some aneurysms of the anterior communicating artery project forwards and downwards and some upwards and backwards. The first type can be exposed by

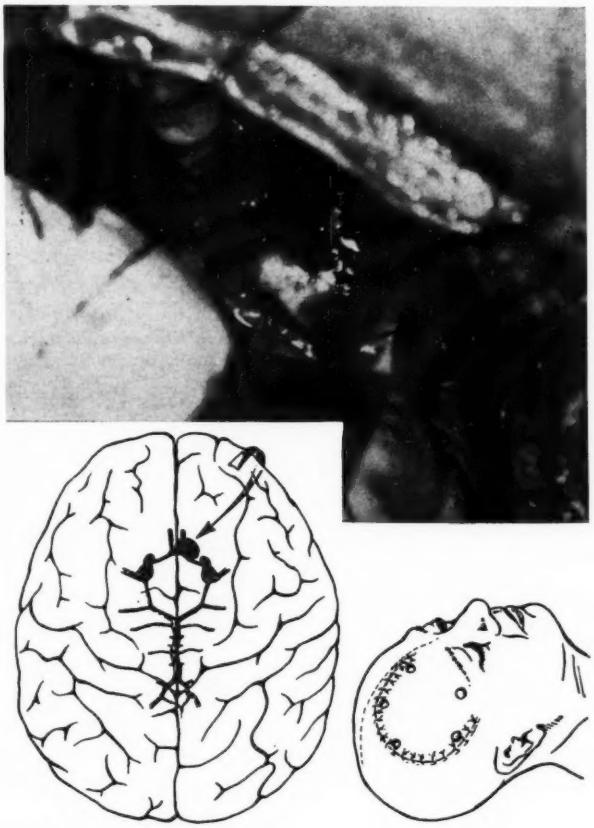

FIG. 4. In this photograph of an operation the aneurysm with the ligature on the neck is clearly seen localized above the chiasma and optic nerves. The drawings indicate the approach.

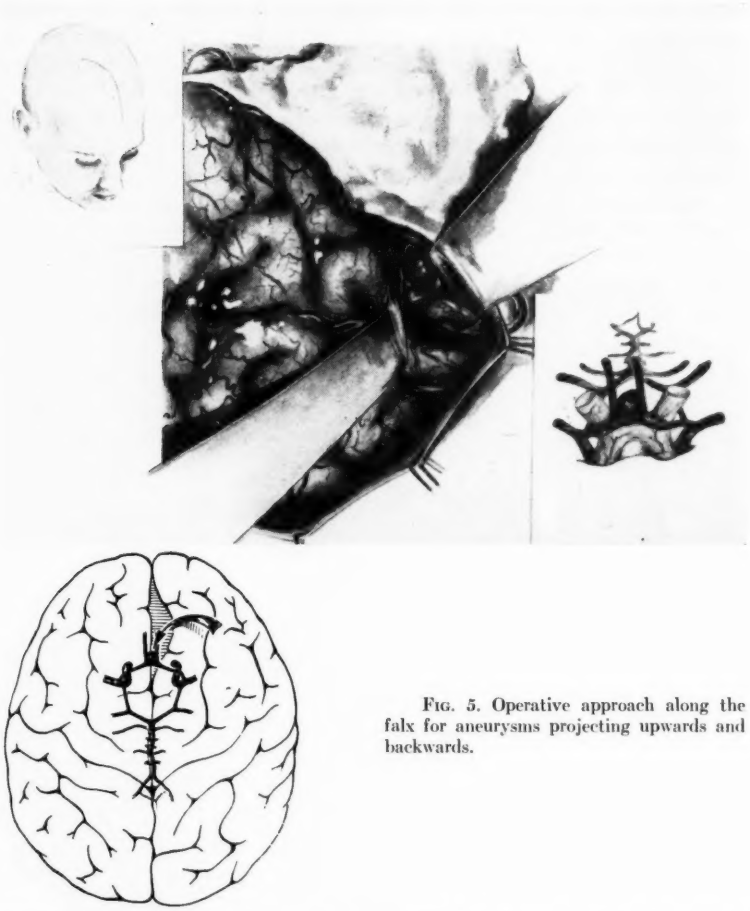

FIG. 5. Operative approach along the falx for aneurysms projecting upwards and backwards.

a subfrontal approach (Fig. 4), which was used in 6 cases. In the second type it is necessary to use a vertical approach down along the falx as described by Tönnis.[31] This approach was used in 10 cases (Fig. 5). Occasionally exceptionally large veins running to the sagittal sinus may be encountered anteriorly, and these must be ligated. This may cause swelling of the frontal lobe and in 1 of our cases re-operation became necessary for resection of the edematous frontal lobe. In some cases, therefore, resection of the frontal pole has been performed at once to avoid the risk of subsequent edema, a factor which might tip the balance in a critically ill patient. This was done in 6 of our 22 intracranial procedures without persisting frontal lobe symptoms. One patient was euphoric for about 2 weeks and another slightly apathetic 5 months later. On the other hand, it was found that with ligation of veins

and retraction of the frontal lobes, 1 of our patients had marked symptoms of impaired function of the frontal lobes, especially of the memory functions. Whether a small resection of the right frontal pole causes less damage than ligation of parasagittal veins on the right side and retraction of frontal lobes has not yet been decided. This question is being investigated by our psychiatrist.

The procedure employed in relationship to the aneurysm itself again must depend upon the operative findings. It is our opinion that direct clipping of the neck of the aneurysm or ligating it with a thread[30] (Fig. 6) is the procedure of choice, although naturally with an aneurysm firmly bound by adhesions this is more hazardous than packing about the sac with muscle and/or gelfoam, and the latter procedure may be the more prudent in some cases. The objection has been raised that dissection of the neck of the aneurysm carries a great risk of rupture during the procedure. This occurred in 2 of our 22 cases of intracranial operation and both patients survived. We have found the use of controlled hypotension by hexamethonium compounds of extreme value in the handling of bleeding in all cases of aneurysm and especially in those in which rupture occurred. Direct ligation or clipping of the neck was performed in 15 of our cases with only 1 fatality in a patient who was comatose and in shock at the onset of the procedure. Jefferson, in discussing a recent paper of Norlén,[25] raised the question of how many aneurysms truly have a neck that is approachable and how many are merely arteriographic interpretations of a "neck" that does not exist. It is interesting to note that in this series the predominance was rather the other way—i.e., in 5 cases in which arteriography had disclosed no neck or only a very slight narrowing it was possible by careful operative dissection to free a neck and occlude it. In all other cases except 3, a neck was found and occluded when the arteriogram indicated one (Fig. 7). In 2 of these 3 cases gelfoam was packed about the sac since it was felt hazardous to attempt dissection because of dense adhesions. In the third, exploration did not reveal the aneurysm itself and no procedure was performed. It should be noted that in 1 of the cases in which gelfoam was packed about the sac, the patient died 4 weeks later from a recurrence of subarachnoid hemorrhage. In this case, gelfoam packing was obviously not protective.

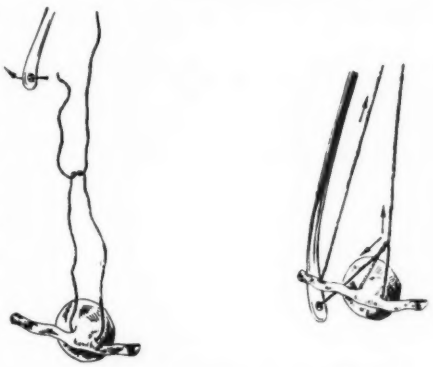

FIG. 6. The technique of ligation of the neck of the aneurysm used by one of us (G.N.). The instrument and one end of the thread is held with the left hand and the tying is then performed by pulling the other end of the thread with the right hand.

The packing of muscle about the aneurysmal sac as early advised by

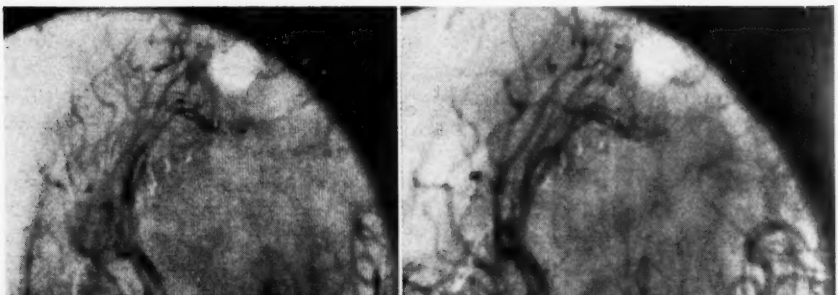

FIG. 7. Pre- and postoperative angiograms of the case shown in Fig. 4. Note the preservation of circulation through the anterior communicating artery after the ligation.

Dott does not strike us as entirely secure either, although Falconer[11] seems to prefer it as a safer procedure, adding a gauze wrapping to his muscle bolster. He performed a direct approach in 8 out of the 11 cases of anterior communicating aneurysm with 6 good recoveries, 1 recovery with mental impairment (in which the anterior cerebral artery was clipped), and 1 death from recurrent bleeding. The only patient who showed recurrence of bleeding and died had had muscle wrapped about the aneurysm. Dandy[4] was opposed to relying on muscle for support. Certainly, as Elvidge and Feindel[9] have re-emphasized, a muscle protection can hardly be expected safely to resist the pressure in an aneurysm when one considers the work of Horsley,[15] who placed the limit of safety of muscle tamponade at about 60–80 mm. Hg. Another factor that to us mitigates against the safety of muscle is the thinness of the aneurysmal sac itself. Forbus,[12] Ectors,[8] Hermann and Macgregor,[14] and Elvidge and Feindel[9] have all emphasized the lack of muscle and elastic tissue in the wall of the sac, the last pointing out that such a wall is rather hazardously supported by muscle packing alone. This doubt has not only been confirmed clinically by Falconer's above-mentioned cases, but by Jefferson,[17] who reported a case of anterior cerebral artery aneurysm packed with muscle, with symptomatic improvement but fatal rupture 18 months later. In another communication[18] he referred to 2 other cases; in one, muscle packed around an aneurysm resulted in a fatal rupture 1 month later, and in the other, carotid ligation was performed 2 months after muscle packing because of continuing pain, which to him indicated activity of the aneurysm. Muscle packing about the aneurysmal sac was performed in only 2 of our cases; both patients have thus far had a satisfactory result. Of 2

patients who had gelfoam packing, 1 died from new bleeding 4 weeks later.

Complete resection of the aneurysmal sac is often advisable when it has enlarged to the point of causing local pressure symptoms. This procedure is, however, probably the most hazardous. Russel[29] reported on a complete excision of an aneurysm in this region by Cone. This was done in 1 of our cases with good result. The danger and difficulty of the procedure in most instances are its main drawbacks.

Trapping of the aneurysm by occlusion of the communicating artery from the circulation is a satisfactory procedure provided that full angiographic studies have demonstrated that no anomalies of the circle of Willis exist and that each carotid is able to take care of the blood flow to its own hemisphere without the aid of collateral circulation from the opposite side. Indeed, this procedure may then be safer and more certain, and it is particularly of value when no aneurysmal neck exists. It is, however, not without its dangers. In Elvidge and Feindel's[9] case of anterior communicating aneurysm trapping was performed with 6 clips on the aneurysm, anterior communicating, and anterior cerebral. The resultant clinical defects, seizures, and evidence of frontal lobe damage in their case point out the inadvisability of sacrificing an anterior cerebral. Trapping was done in only 1 of the present series, a case in which the aneurysm represented a diffuse dilatation of the anterior communicating artery with no neck whatsoever. This patient was critically ill at operation and died the following day. This single case is not, of course, interpreted as indicative of the value of the procedure.

RESULTS

In our 26 cases of anterior communicating artery aneurysms (Table 1) 24 were surgically treated, with 2 operative fatalities, and 1 subsequent death from subarachnoid hemorrhage in a case in which gelfoam had been packed about the aneurysmal sac. One other patient died from subarachnoid hemorrhage $1\frac{1}{2}$ years later in a case early in the clinic experience, in which an exploratory craniotomy but no surgical treatment of the aneurysm had been performed.

The time that has elapsed since operation is in most of these cases too short to permit any conclusions regarding the late results. Of the 15 patients who were treated by occlusion of the neck of the aneurysm, 14 survived the operation. None of these 14 patients showed any sign of recurrence of bleeding. Five returned to work and 2 of these had had impaired vision which improved considerably after the operation. Five left the hospital without any neurological signs and are well but have not yet started to work though they are supposed to do so. Of 2 patients with hemiplegia before operation, 1 is improving and 1 is unchanged. In 1 case, hemiplegia appeared some days after operation, probably as a result of thrombosis; some improvement has occurred, but the patient is still hemiparetic. In 1 case, as previously mentioned, there was pronounced postoperative impairment of memory function; some improvement has occurred.

We have tabulated the time interval from bleeding to operation, the condition of the patient at operation, and the operative mortality in Table 3. Our series is too small to draw any logical conclusion and is weighted as to time in that not more than 6 patients were operated on during the first

TABLE 3

Time interval from bleeding to operation and condition at operation compared with operative mortality in 22 cases of subarachnoid hemorrhage surgically treated

Interval from Bleeding to Operation	No. of Cases	Condition at Operation			
		Comatose	Subcomatose	Operative Risk*	Operative Death
Up to 6 days	1	+		5	1
	1		+	4	
	1		+	4	
	1		+	3	
	4				1
1½–3 weeks	1	+		5	
	1		+	2	
	2				0
1–3 months	1		+	5	1
	3			1	
	3			2	
	2			3	
	9				1
Over 3 months	4			1	
	2			2	
	1			3	
	7				0
Total	22				2

* Classed 1 to 5, with 1 a perfect operative risk and 5 the poorest operative risk.

3 weeks after hemorrhage, because of problems of transportation and bed capacity. It is included here only to record the data so that others in the future may add them to their experience in attempting to answer this difficult question of when to operate and when to wait. The patients who survived for longer intervals were, as would be expected, in better condition and did well. How many patients might have been referred had they survived conservative management we cannot say (nor, in fairness can we say how many would have died from early surgical intervention). It is an important problem which needs clarification.

In summary then, it would be our conclusion that a direct intracranial approach with ligation of the neck of the aneurysm (when possible) is the

procedure of choice for aneurysms in this specific location. The other procedures, especially trapping, have their indications and contraindications, but on the whole we would echo the words of Dr. Rudolph Jaeger of Pennsylvania who in discussing a paper of Hamby[13] remarked, "The aneurysm must be attacked directly and tied off. This is the only specific treatment for an intracranial aneurysm."

SUMMARY

1. Because of their location on the major vessel for collateral circulation in the anterior part of the circle of Willis, it is felt that aneurysms of the anterior communicating artery present certain specific problems in management different from other intracranial aneurysms.

2. A series of 26 cases of anterior communicating artery aneurysm is presented, this representing the entire unselected material of this category from this clinic up to April 1952.

3. With the experience in this series as a background, the problems of incidence, etiology, symptomatology, and management of this type of case are discussed. Special attention is drawn to vasospasm in relation to subarachnoid hemorrhage and its clinical importance.

4. The need for complete angiographic evaluation of all cases prior to surgical intervention is stressed.

5. Because of the poor outlook of conservative therapy, it is concluded that these aneurysms are best handled by a direct surgical approach. It is concluded that when possible a direct attack on this type of aneurysm with occlusion of its neck by clip or ligature is the procedure of choice. The optimum time for surgery following the hemorrhage is discussed.

6. In this series of 24 surgical cases only 2 could be considered as operative deaths. In addition there were 2 subsequent deaths from subarachnoid hemorrhage ascribed to what we consider inadequate procedure.

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SURGICAL TREATMENT OF DEAFNESS FOLLOWING MENINGITIS*

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GENERALLY it is assumed that, when a child has become deaf or hard of hearing after meningitis, little if anything can be done to help the hearing. A review of the literature has not provided any optimistic information on the subject. Davis[1] wrote that, "The knowledge of the nature of the injury in meningitis and in other forms of nerve deafness allow us to state categorically that *no type of treatment, medication, or stimulation will improve the hearing of anyone whose deafness is due to meningitis or for whom a diagnosis of true nerve deafness can be made.*"

Experience with 6 cases of this type of deafness treated surgically has shown that some patients can be helped by radical measures directed to the 8th cranial nerve.

The first patient in this small series was actually not admitted for treatment of his deafness and it was a surprise to find that something might perhaps be done to help his hearing.

Case 1. J.W., aged 5 years. The presenting complaint was convulsions following meningitis and in the investigation an air encephalogram was done. The films showed poor filling of subarachnoid channels over the cerebral hemispheres. In the next few days hearing was much improved and it was noticed that the boy's balance was disturbed during the time that hearing was better. This effect on hearing and balance lasted for about 10 days. Apparently the 8th nerves were able to function better for a time and this temporary change was attributed to the mechanical effect of the air in the subarachnoid space around the 8th nerves. Accordingly an operation was planned in which the basal cisterns and cerebellopontine angles could be explored.

Operation. On Jan. 31, 1947 suboccipital craniotomy was done. Anesthetic was ether by nasal catheter.

With patient face-down on the cerebellar head-rest, a curved incision was used from one mastoid tip to the other. After reflecting muscles and periosteum from occipital bone down to the arch of the atlas, bone was removed from each occipital squama laterally as far as the lateral sinus. The bone removal was carried down into the digastric groove and each oval-shaped opening measured about 3.5 by 5.0 cm. (Fig. 1).

The foramen magnum was unusually large. When the dura mater was opened

* Presented in April, 1951 at the nineteenth annual meeting of the Harvey Cushing Society in Hollywood, Florida.

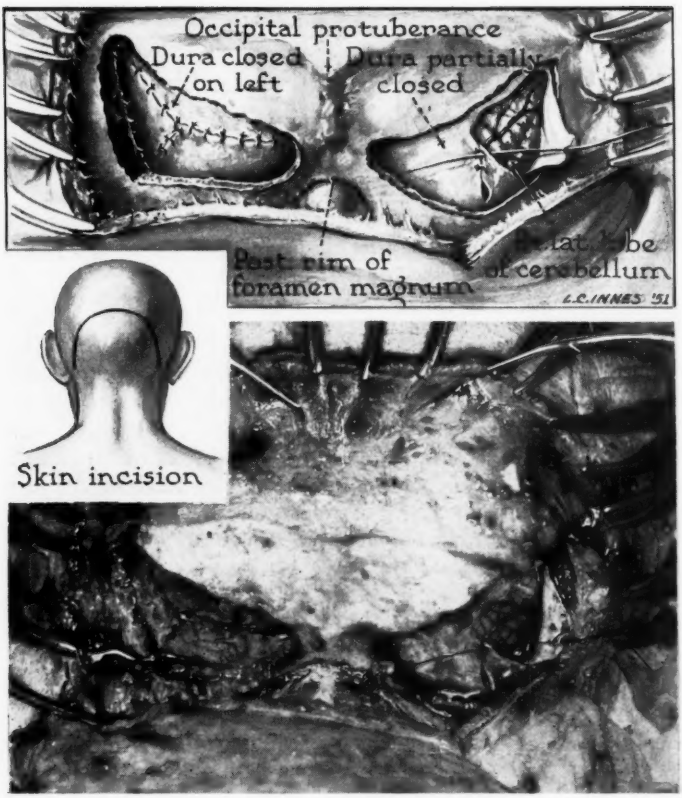

FIG. 1. *Case 1.* (*Above*) Drawing of the general operative exposure. (*Below*) Photograph showing the two openings in the bone; the dura mater is closed on the left, and partially closed on the right with the cerebellum still in view above. It is evident that this type of exposure uncovers more bone than is necessary since it did not appear indicated to expose the cisterna magna more widely.

the cerebellum bulged until the cisterna magna had been opened. The arachnoid over the latter was quite tough and the cisterna magna was serrated at its edge, suggesting atrophy of the cerebellar folia. The left cerebellopontine angle was explored first and as the cerebellum was retracted medially the left auditory nerve was found to be covered with thickened, milky arachnoid. The anterior inferior cerebellar artery was folded over the nerve and then formed a second fold down in front of it before turning back again toward the cerebellum. This artery was quite beaded, apparently from traction by the bands of adhesions between cerebellum and dura mater (Fig. 2).

The adhesions were freed by opening the arachnoid widely and a small artery passing from the anterior inferior cerebellar artery toward the internal meatus was coagulated and divided. The folds of arachnoid could not be torn with the small

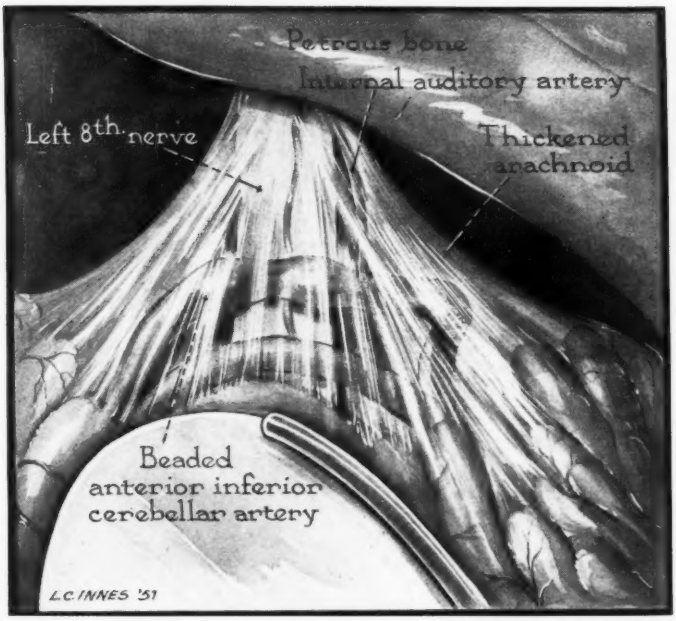

FIG. 2. *Case 1.* The left 8th nerve as it was first exposed, obscured by the thickened and white arachnoid. The beaded effect can be seen on the anterior inferior cerebellar artery. (This drawing and that in Fig. 3 were elaborated from careful sketches by Dr. Robert S. Knighton* who was resident in neurosurgery and assisted at the operation.)

dissector and had to be cut with a scalpel or the cutting cautery. By then the beading on the anterior inferior cerebellar artery was appreciably less (Fig. 3). The work was facilitated by the use of a magnifying loupe. The 8th nerve appeared normal.

The right 8th nerve was exposed in the same way. Arachnoid was less thickened than on the left although it could not be torn with a small dissector (Fig. 4). A small artery passing from the anterior inferior cerebellar artery to the petrous bone was coagulated and divided as it seemed to be holding the larger artery against the 8th nerve. The 5th nerve was not seen on either side but a good view was obtained of the 9th, 10th and 11th nerves. The cerebellum appeared normal with the exception of the suspected atrophy around the cisterna magna.

The wound was closed in layers with black silk and without drainage.

Postoperative Course. Two days after operation the patient could hear fairly well. On the 4th day talking was improved and he seemed more cooperative and restful. He was noticing new sounds; for example, he asked his father about the sound of a siren which he had heard for the first time. Temperature varied from 99.8 to 102° by rectum and was normal on the day of discharge from hospital, 7 days after operation. There had been no convulsions since the day he was first admitted.

* Now neurosurgeon at Henry Ford Hospital, Detroit, Michigan.

FIG. 3. *Case 1.* The left 8th nerve after it was cleared of the thickened arachnoid. The beading on the artery was appreciably less than when first seen.

Subsequent Course. A letter from the patient's mother in March, 1947 indicated that her son was improving steadily and that his hearing was very much better. He was able to repeat the alphabet and to count up to ten. However, the minor spells were returning and more recently they were becoming worse. They were ushered in by epigastric discomfort.

One year after operation the hearing was apparently normal. The parents reported that, if they called from a distant part of the house, the boy would seem to hear them readily.

On July 26, 1948, 18 months after operation, hearing still seemed good. Audiogram showed loss of between 30 and 60 decibels on the right and from 30 to 50 on the left (Fig. 5).

In the next 5 years, 5 other patients were treated along the same lines. This makes a series of 6 cases, in 5 of which a craniotomy was performed. The clinical material will be tabulated in brief case reports arranged chronologically in a later section of this paper.

In all 6 cases the impaired hearing or deafness began during the acute illness. The organism was meningococcus in 2 cases, pneumococcus in 3 and staphylococcus in 1. Patients were from $4\frac{1}{2}$ months to 19 years of age at the time of the acute infection. It was then from 3 to $7\frac{1}{2}$ years before they had the operations reported here. In the 3 cases in which the patients were

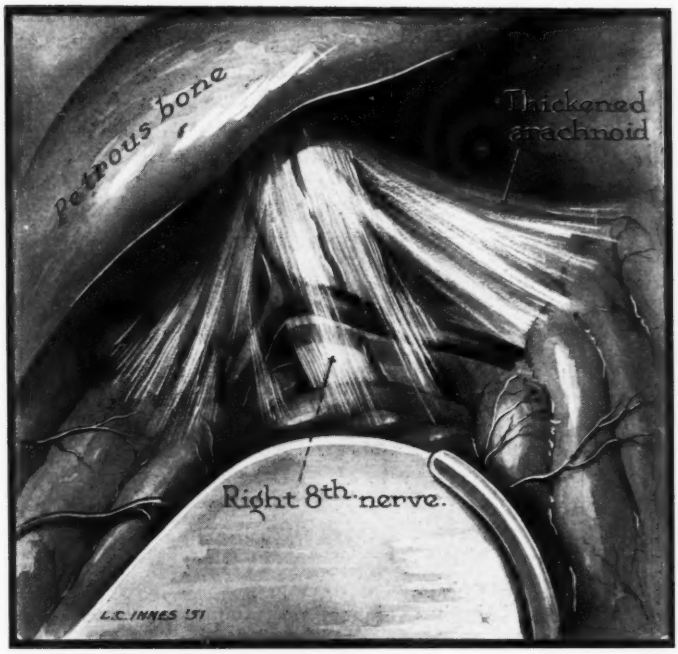

FIG. 4. *Case 1.* View of the right side. The arachnoid is less thickened than on the left side. As mentioned in the text, the internal auditory artery passing from the anterior inferior cerebellar artery to the internal meatus seemed to be distorting the vessel and pulling it against the nerve. It was divided after being electrocoagulated.

helped, the time interval from the acute illness to the operative treatment was 3 years, $4\frac{1}{2}$ years, and $7\frac{1}{2}$ years respectively.

Hearing ranged from greatly impaired to completely absent. No patient was helped by operation who was completely deaf from meningitis.

In all cases an air encephalogram was done as a preliminary step with the

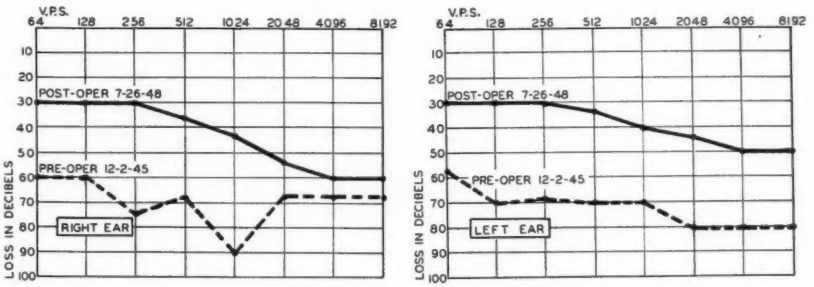

FIG. 5. *Case 1.* Audiogram showing the comparative values in each ear, before and after operation.

hope of gaining information about the subarachnoid cisterns around the base of the brain and the 8th nerves. Also it was used as a test to see if function of the 8th nerve would be altered by the mechanical effect of the air. This might be evident in a temporary or permanent effect on hearing or on control of balance. In cases of postmeningitic deafness, when the air encephalogram did not have some, even temporary, effect on 8th nerve function, there was no improvement following operation to explore the 8th nerves directly.*

OPERATIVE TECHNIC

For infants the anesthetic was ether with the usual pre-medication. Pentothal sodium was used in patients over 7 years of age. One per cent novocain was injected in skin and muscle. For the bilateral operation the patient was in face-down position but was not intubated. There was no mortality.

Incision in all but Case 1 was of small hockey-stick shape behind the mastoid. Bone was removed to uncover the outer limit of the cerebellum and usually it was not necessary to reach the cisterna magna for drainage of subarachnoid fluid.

After retracting the cerebellum medially a magnifying loupe was found to be helpful. Arachnoidal bands or septa could then be divided with the pointed scalpel or cautery-tip without injury to adjacent blood vessels. Small arteries and any bridging veins between cerebellum and petrous bone were electrocoagulated and divided, when necessary. Care was taken never to push the retractor against the 8th nerve.

In Cases 3 and 4 the nerve sheath was split lengthwise without any surface bleeding. From the present study one cannot say that it was helpful to do this.

Facial weakness, even slight and transient, did not occur in any case.

When the anterior inferior cerebellar artery was pressing on the 8th nerve it was from below, so that it was pushing against the cochlear portion. In this event the artery was dissected free enough to move it away from the 8th nerve. If necessary, a small piece of gelfoam can be interposed.†

OPERATIVE FINDINGS

Lesions affecting the 8th nerve, as found at operation in the present series, will be considered under three categories: (1) Changes in arachnoid. (2) Abnormalities in 8th nerve. (3) Compression by adjacent artery.

1. *Changes in Arachnoid.* Milky appearance of arachnoid was found in most cases following meningitis, indicating cellular infiltration or proliferation. With this there was thickening of the membrane so that it usually

* It is obvious that this series is too small for a conclusion on this point.

† N.B. In an adult with Ménière's disease the vestibular portion of the left 8th nerve was divided. The patient also had poor hearing on the left and tinnitus. Gelfoam was used to hold the artery away from the nerve and, 3 months later, the patient was not only free from vertiginous attacks, but her hearing was normal and tinnitus occurred only if she would lie on her left side for a long time and not always then.

could not be torn but had to be cut with scissors, pointed knife or cautery. Such bands of thickened arachnoid were presumably sufficient in Case 1 to give the adjacent artery a beaded appearance. Traction by constricting bands could produce mechanical compression of the 8th nerve and also could embarrass the blood flow, both arterial and venous, in the nerve.

In Case 6 there was obvious loculation so that a series of sacs of fluid seemed to stretch the 8th nerves. After the sacs had been opened widely no investment of arachnoid could then be found on the nerves, indicating that the lesion was not a multilocular subdural hydroma.

2. *Abnormalities in the 8th Nerve.* In Case 4, the 8th nerve was bluish at first and more pink after the arachnoidal tension on it had apparently been relieved. Surface vessels, not visible at first on the nerve, were seen to open up when the arachnoidal bands were freed. In Case 3, the left nerve was discolored an ivory yellow and it was swollen.

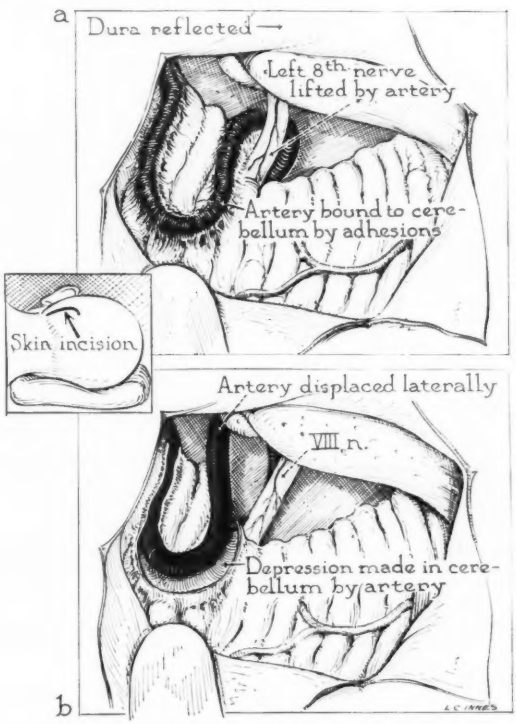

FIG. 6. *Case 2.* This represents the operative appearance on the left side. In (a) one can see the anterior inferior cerebellar artery bound tightly to the cerebellum by thickened arachnoid and adhesions. The artery pushes the 8th nerve up and forward, pressing chiefly against the cochlear portion. In (b) the artery has been dissected away from the cerebellum. Then it was pushed down where a small protrusion on the petrous bone held it away from the 8th nerve which was then straight.

3. *Compression by Adjacent Artery.* In Case 2, only the left nerve was exposed. The anterior inferior cerebellar artery was bound to the cerebellum by heavy adhesions and was lifting the 8th nerve from behind (Fig. 6). This would exert the greatest pressure on the cochlear portion of the nerve, with consequent effect on hearing.

RESULTS OF AIR ENCEPHALOGRAPHY

Of 6 patients who had air encephalography, 3 had improved hearing following it. In Case 1, the improvement was temporary. In Cases 2 and 5, it was definite but not enough to be considered satisfactory improvement. In 2 of these 3 cases operative exposure of the 8th nerves gave additional and more lasting benefit. The third patient has had air encephalography only. All 3 patients had had meningitis but 2 had at least a little hearing when first seen.

Sense of balance was affected in 2 patients, Cases 1 and 5, as a result of the air injection. It was transient, lasting for a few days and 3 weeks respectively.

RESULTS OF OPERATION

In the 5 patients operated upon improvement in hearing varied from none to very good. The best results were in patients who had slight or temporary benefit from the air encephalography. Two patients had useful return of hearing after operation.

Some improvement in balance control was found in 1 case, pointing to partial recovery of the vestibular portion of the nerve. This patient was Case 4, who had no change in hearing as a result of operation.

The measures that seemed to help the nerve were: (1) Release of subdural fluid. (2) Opening of cystic subarachnoid formations. (3) Dissection of thickened bands of arachnoid from around the 8th nerve. (4) Mobilization of anterior inferior cerebellar artery when it was pressing on the nerve.

TABULATION OF CASES

Case 1. J.W. Age 5 years Admitted Nov. 19 1946

Etiology of deafness—meningitis—at 4½ months following right otitis media
staphylococcus aureus hemolyticus

Hearing—obviously decreased after the meningitis
at 9 months, would jump at a loud noise
later, could not hear other children when playing
at 5 years, it seemed possible that a loud tuning-fork was heard on the right
audiogram showed loss of from 60 to 90 decibels on right and from 56 to 80 decibels on left

Balance—not disturbed

Mentality—subnormal
had frequent temper tantrums
co-operated poorly, would spit at examiner

Speech—began at 28 months
always poor, as though tongue were thick
at 5 years, was still using single words only

Air encephalogram—films showed—poor filling of subarachnoid channels over the hemispheres
enlarged cisterns around cerebellum

general effect—patient not unduly sick
effect on hearing—hearing was better within 5 days and improved for another week. Then, within a few days, it regressed to the original level
effect on balance—between the 5th and 8th days patient staggered much and indicated that he felt dizzy

Operation—bilateral
findings—arachnoid thickened and white, more on left than right
left anterior inferior cerebellar artery was beaded by bands of arachnoid, less so after the bands were divided (Figs. 1, 2, 3)
procedure—bands of arachnoid cleared away, including small vessels
result—hearing improved in 2 days. In 4 days patient heard a siren for the first time. One year after operation his parents felt that, for practical purposes, his hearing was normal

Case 2. J.R. Age 9 years Admitted Sept. 27, 1948

Etiology of deafness—meningitis—at 20 months
meningococcus
Hearing—almost absent on left although loud tuning-fork heard at 1½ inches
school report showed decrease to 27 per cent of normal on right and absent on left
Weber referred to right side
Balance—not disturbed
Mentality—not too alert
attended school but progress said to be fair
Speech—enunciation poor
would make short sentences
Air encephalogram—x-rays—poor filling of subarachnoid channels
general effect—not very sick, went home in 2 days
effect on hearing—patient said that after 1 day he could hear better so that a tuning-fork could be heard on the left better than on the right in the Weber test sound was referred to the left
effect on balance—none
Operation—left side only
findings—arachnoid somewhat tougher than usual
anterior inferior cerebellar artery was pressing against cochlear division in two places and was bound to the cerebellum (Fig. 6)
procedure—artery was mobilized and was moved away from the 8th nerve
result—slow but steady improvement in hearing
1 year after operation a hearing aid was provided on the right
3 years after operation, patient could hear the examiner's spoken voice without a hearing aid. Patient's mother reported that he was not turning up the radio as loud as a few months before

Case 3. C.Y. Age 4½ years Admitted Oct. 18, 1948

Etiology of deafness—meningitis—at 7½ months following otitis media
pneumococcus
Hearing—not very good
Mentality—much retarded
had not been trained for the toilet
generally irritable, unco-operative
would pound head and hold it as though it hurt
Speech—no words at all
Air study—ventriculogram showed ventricles greatly enlarged.
no air entered the basal cisterns
Operation—bilateral
findings—arachnoid very tough and thickened on both sides
left 8th nerve was an ivory-yellow color, also somewhat swollen
there appeared to be two layers of arachnoid that required opening before the left nerve was seen
procedure—thickened arachnoid was cleared away from both 8th nerves
the sheath was split over the left cochlear portion, without bleeding

result—1 year after operation there was only suggestive evidence that patient could hear. He sometimes would turn around when he was called. Also he liked to play with a rattle
4 years after operation the parents felt that the child could not hear. He was less restless and appeared to have less headache
mentally he was only a little better than before and he had been admitted to an institution

Case 4. E.N. Age 23 years Admitted Aug. 15, 1948

Etiology of deafness—meningitis—at 19 years
meningococcus
Hearing—completely absent after the acute illness
tinnitus on shaking head or when excited
Balance—quite unsteady with eyes closed, only since the acute illness
Mentality—very bright
Speech—excellent but talked in a monotone and low pitch

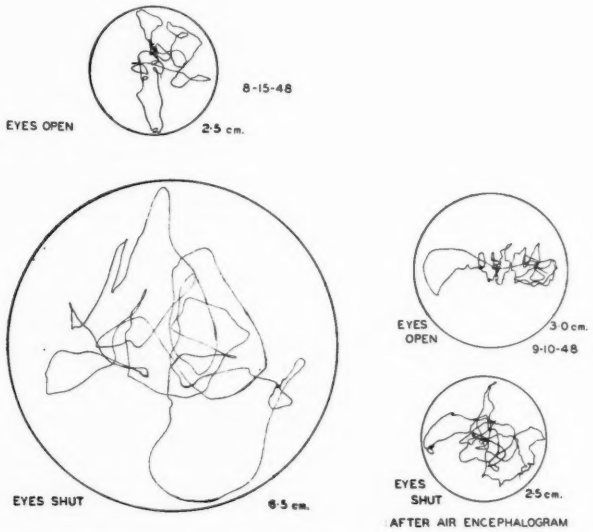

FIG. 7. Romberg tracings in *Case 4*. These are made by placing a small pen on the patient's head and a horizontal board above this so that, in the Romberg test, the patient makes a tracing on the paper. The method has been described previously.[2] The tracings made before operation show the great amount of swaying with eyes closed and the later tracings show the improvement mentioned in the text. The tracings with eyes open are about twice as wide as the average normal.

Air encephalogram—x-rays—normal filling of subarachnoid spaces over convexity and in basal cisterns
general effect—not much upset
effect on hearing—no change of any kind
effect on balance—less unsteadiness with eyes open and much less unsteadiness with eyes shut (Fig. 7)
Operation—left side only
findings—arachnoid tough and adherent to the anterior inferior cerebellar artery and to the 8th nerve
8th nerve was quite blanched at first but after the arachnoidal adhesions had been cleared away it was pink and small vessels on the sheath were more numerous

procedure—arachnoid was cleared away from the artery and nerve sheath was split without bleeding
result—no change whatever

Case 5. E. I. Age 8 years Admitted Oct. 21, 1948

Etiology of deafness—meningitis—at 5 years
pneumococcus
Hearing—was lost during the acute illness
no demonstrable hearing when tested by audiometer
Balance—very dizzy for 3 or 4 months after the meningitis
Mentality—apparently normal
Speech—good
Air encephalogram—x-rays—no filling of subarachnoid channels over the surface
right lateral ventricle was filled and of normal size
left lateral ventricle did not show
basal cisterns well filled
general effect—not particularly upset
effect on hearing—no immediate change
8 months later patient's mother felt that there had been no change although patient was able to hear a tuning-fork by air conduction on the right and in the Weber test the sound was referred to the right. At school the teachers thought that hearing was present
in the next 3 years there was no additional improvement in hearing
effect on balance—for about 3 weeks after the air encephalogram patient was quite unsteady

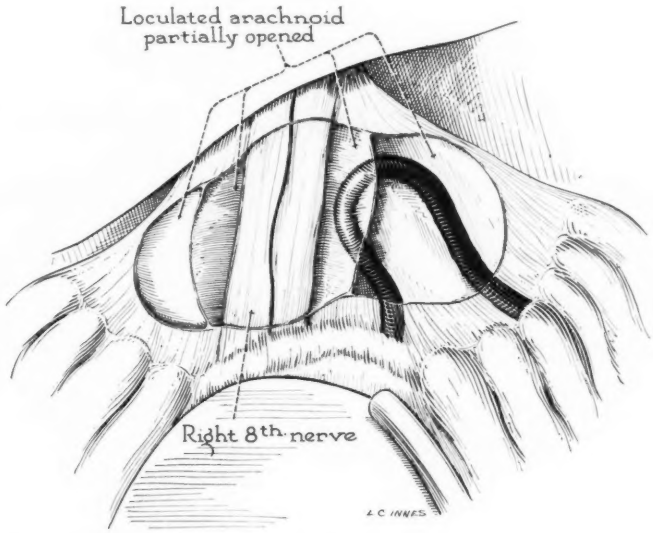

FIG. 8. *Case 6.* The cystic formation of the arachnoid is depicted on the right side where septa were found so that when the arachnoid was first opened down toward the 9th nerve, it was still necessary to open it in two or three other places and release additional fluid before the 8th nerve was fully exposed. The 8th nerve appeared normal here although an unusual extent of it was visible, suggesting that the loculations in the arachnoid had stretched the nerve.

Case 6. G. R. Age 8 years Admitted Dec. 9, 1950

Etiology of deafness—meningitis—at 2½ years
pneumococcus
Hearing—not completely lost on left but not good enough to use a hearing aid
Balance—not very steady on his feet
Mentality—good
Speech—not very good, attributed to inadequate teaching
Air encephalogram—x-rays—no air in subarachnoid space over the hemispheres
large subdural crescents
general effect—very sick for several days
effect on hearing—none
effect on balance—none
Operation—bilateral
findings—subarachnoid space appeared to have several compartments so that it was loculated and the space between the petrous bone and cerebellum was increased so that the 8th nerves appeared stretched (Fig. 8)
procedure—arachnoid was opened widely
result—disposition improved in that patient was less irritable
hearing not apparently altered although patient was trying more to talk and the parents thought he might be hearing some sounds better

SUMMARY

Six cases are reported of children who were hard of hearing or totally deaf following meningitis between 3 and 7 years previously.

Operative exposure of the 8th nerves allowed release of arachnoidal adhesions, loculated fluid collections or of compression by the adjacent anterior inferior cerebellar artery.

In 2 of the 5 patients operated upon there was improvement in hearing.

In 3 cases air encephalography was followed by temporary or slight improvement in hearing. This diagnostic measure was used to try to decide on the advisability of operation. The 2 patients who were helped by the operation had had some improvement after the air injection.

I am indebted to Dr. Edward C. Oberc, of Pasadena, who assisted in the care of most of the patients, to Helen Kennedy, Ph.D., Professor of Education in Los Angeles State College, for studies of hearing in several of the cases, and to Mrs. L. C. Innes for her painstaking drawings supplemented by studies in the anatomy laboratory and at the operating table.

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CASE REPORTS

NONCHROMAFFIN PARAGANGLIOMA OF THE JUGULAR FORAMEN

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This paper deals with a carotid-body-like tumor found in the jugular foramen, similar to the tumors recently described by Lattes[12] as nonchromaffin paragangliomas.

Kohn,[10] in 1900, classified the carotid body as a paraganglion, in the belief that it was of sympathetic origin and part of the chromaffin system.

Small masses of similar paraganglionic tissue have since been described in the following locations:

1) *At the level of the middle ear.* As early as 1840, Valentin[26] described a *ganglíolum tympanicum*, associated with the tympanic branch of the 9th cranial nerve. Later, in 1879, Krause[11] showed that this structure resembles the carotid body and this has been confirmed recently by the studies of Guild[8] and Lattes and Waltner.[13] In addition to the paraganglion tympanicum, Guild[8] described what he called the glomus jugularis "in the adventitia of the dome of the jugular bulb, immediately below the bony floor of the middle ear."

2) *At the aortic-pulmonary region.* Paraganglia related to the major vessels at the base of the heart were mentioned in 1902 by Biedl and Wiesel.[2] In the middle thirties, Palme,[21] Seto,[24] Muratori[19] and Nonidez[20] described these structures in detail. From their contributions, well summarized by Boyd,[4] we learn that these "aortic bodies" are similar to the carotid bodies and that they can be found in four different locations: a) above the ductus arteriosus; b) on the trunk of the pulmonary artery, near the origin of the left coronary artery; c) near the root of the innominate artery; d) on the left part of the aortic arch.

3) *Within the ganglion nodosum.* Muratori[18] described in 1932 a paraganglionic formation inside the vagus nerve, at the level of the ganglion nodosum, and called it *paragangli intravagali.* He found it in different species of the bird, "Ucelli." His findings were confirmed by the works of White[28] and Lattes,[12] who described the paraganglion intravagale in man.

4) *In the vicinity of the ciliary ganglion.* Botár and Pribék[3] and Gosses[7] have described a paraganglionic formation related to the ciliary ganglion of monkeys. This structure has not been described in man.

Although serious doubts have been raised as to the sympathetic origin of the carotid body and related structures,[4,22] the term paraganglion has continued to appear in the literature. In 1926 and 1928 de Castro[5,6] produced good evidences questioning the chromaffin reaction in the cells of the carotid body. Hollinshead[9] in

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1940 made a review of the subject, arriving at the conclusion that the carotid bodies and cardio-aortic bodies are nonchromaffin in nature. Watzka[27] classified these bodies as "nonchromaffin and nonepinephrine producing paraganglia."

Accordingly, tumors related to these structures have been most often described as paragangliomas. Lattes and Waltner[13] suggested that all these tumors originating from the carotid body or related structures be designated nonchromaffin paragangliomas, stressing that "the modifying adjective 'nonchromaffin' would help to avoid confusing them with the chromaffin paraganglioma or pheochromocytoma."

Nonchromaffin paragangliomas have been described in association with the carotid body, aortic bodies, glomus jugularis and paraganglion intravagale.

Carotid-body-like tumors have been recognized and described in the literature since 1891.[14] Recently, Monro[16] surveyed 223 reported cases which he considered acceptable as carotid-body tumors and added 5 more cases to the literature.

Carotid-body-like tumors originating away from the bifurcation of the common carotid artery are a relatively recent discovery. Stout,[25] in 1935, was the first to report such a tumor, when he described a neoplasm "developed in the course of the vagus nerve, in connection with the ganglion nodosum, . . . morphologically a paraganglioma."

Rosenwasser,[23] in 1945, described a "carotid body tumor of the middle ear and mastoid" and suggested its origin from the glomus jugularis. Further reference to carotid-body-like tumors of the glomus jugularis can be found in the paper of Lattes and Waltner,[13] in which they reviewed the literature and contributed 8 new cases. Isolated cases have been reported since their paper, giving a total of 28 cases.

Lattes,[12] in 1950, reported the first two examples of such tumors occurring in the vicinity of the aortic arch and suggested that they originated from the aortic bodies. One more case has since been added by Monro.[17]

After the original case of a paraganglioma of the ganglion nodosum reported by Stout, there appeared in the literature only one paper reporting such tumors. Lattes,[12] in addition to contributing the first 2 cases of nonchromaffin paragangliomas of the aortic bodies, presented a follow-up study on Stout's case together with 2 new cases of nonchromaffin paragangliomas of the ganglion nodosum. It is interesting to observe that in his 2 new cases the paragangliomas were multicentric in origin: one involved the ganglion nodosum on one side and the carotid body on the opposite side; the other "exhibited three independent but histologically identical growths originating respectively in the vagus nerve at its exit from the base of the skull, at the bifurcation of the common carotid artery and in the adventitia of the aortic arch near the obliterated ductus arteriosus." There is another case that should be mentioned. Dr. R. E. Semmes, discussing a paper by Alexander, Beamer and Williams[1] on tumors of the glomus jugularis, stated that he had found a similar tumor blocking the jugular foramen and incorporating the 9th, 10th, 11th and 12th cranial nerves in a 32-year-old woman. Dr. W. H. Mathews[15] told us of his experience with a similar tumor "in the region of the internal and external jugular veins, involving the vagus nerve and whose true extent was not determined though it apparently extended upwards to the base of the skull." This tumor was only partially removed and was diagnosed as a nonchromaffin paraganglioma of the vagus nerve.

REPORT OF OUR CASE

Mrs. R.G., 42-year-old white female, was admitted to The Chicago Memorial Hospital on Jan. 15, 1951, with the chief complaint of continuous vomiting during the previous 5 days.

She had had dull frontal headaches off and on most of her life, easily relieved by aspirin. Otherwise she had always enjoyed good health until 2 months prior to admission, when, for the first time, she began to notice some pains in the head. These pains were of a different character and consisted of an almost continuous feeling of pressure in the back of the head and a sharp pain behind the right ear. More or less at the same time she began to notice strange noises in the right ear, such as roaring and ringing, followed by slight impairment of hearing on that side. Some difficulty with her throat, described as a feeling that the "throat has swollen up " also made its appearance about 2 months prior to admission. These symptoms were followed by occasional vomiting and a change of her voice. Speech at first acquired a nasal quality and this grew progressively worse. During the last 5 days prior to admission she was barely able to whisper. She had three very severe episodes of vomiting, during which she also vomited through the nose. The vomiting lasted several hours on one occasion, all day on another, and had been continuous for 5 days at the time of her admission. She stated that she had noticed during the previous 2 months that her right arm was slightly awkward and that she felt slightly lightheaded every morning.

Examination. She was an acutely ill patient, very dehydrated, vomiting clear or bile-like fluids or else retching continuously. She was very restless and apprehensive. B.P. was 130/80, pulse 90, and respiratory rate 22. Her abdominal muscles were somewhat tender, particularly close to the ribs. This was interpreted as being caused by her continuous vomiting.

The positive neurological findings were: (1) Slight loss of hearing on the right, with air conduction better than bone conduction, indicating a nerve type of deafness. When the tuning fork was applied to the vertex the sound was referred to the left ear. (2) The palate deviated

FIG. 1. Section through jugular foramen, with internal auditory meatus in the background. Jugular foramen is filled with paraganglioma which enmeshes the normal structures.

to the left, with a typical curtain phenomenon. The gag reflex and the sensibility of the palate were decreased on the right side. (3) The right vocal cord was bowed and paralyzed, indicating involvement of the vagus. Phonation was very impaired, the patient only being able to whisper. (4) The right sternocleidomastoid muscle was very weak and atrophic. (5) There were slight dysmetria and dysynergia and slight impairment of rapidly alternating movements of the right arm. There was weakness of the right trapezius muscle which affected the movements of that arm.

Caloric tests were normal. All tendon and superficial reflexes, including corneal reflexes, were normal, except for the gag reflex on the right. Except for the palate and pharynx, as noted above, sensation was normal throughout. There was no nystagmus, no facial weakness, no weakness of any extremity. No lymph nodes or other masses could be palpated in the neck or elsewhere. There was no evidence of any pathology in the nasopharynx.

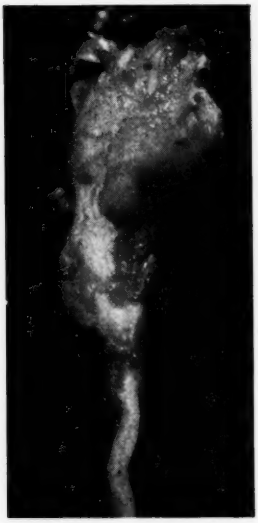

FIG. 2. Tumor mass removed from the jugular foramen. Vagus nerve is seen emerging from lower end of the tumor and lying in front of the jugular vein. From the left side of the tumor mass, a nodule of tumor is seen which protruded into the jugular vein.

Laboratory findings, including the Wassermann and Kahn tests, were within normal limits. Roentgenograms of the chest and skull were normal except for a very slight haziness of the periantral cells of the right mastoid and a lack of pneumatization of the right mastoid tip.

A diagnosis of a tumor in the right jugular foramen was made.

Operation. On Jan. 18, 1951 a right cerebellar craniotomy was performed under intratracheal ether anesthesia. The right 7th, 8th, 9th, 10th, and 11th cranial nerves were exposed. The 7th and 8th appeared normal both in size and color. The 9th, 10th, and 11th nerves, on the other hand, were grayish in color and did not appear at all healthy. However, no tumor could be seen about them or about the jugular foramen. The brain stem and the space anterior to it were visualized and no tumor mass was seen. Just as we were preparing to close the incision, the anesthetist announced that the systolic blood pressure, which had been running from 160 to 180 throughout the procedure, had dropped to 110 mm. of Hg. The glucose solution, which had been running intravenously during the operation, was replaced with blood. Suddenly respiratory and cardiac activities ceased and the blood pressure could not be obtained. In spite of accelerating the administration of blood, giving artificial respiration and stimulants, and forcing oxygen through the intratracheal tube, respiratory and cardiac activities were not resumed.

Autopsy. The brain was entirely normal. A part of the base of the skull, incorporating the right jugular foramen and related structures, was removed. This specimen consisted of the petrous pyramid and the floor of the posterior fossa on the right side. The petrous pyramid itself, the opening over the tip of the petrous pyramid into Meckel's cave, the internal auditory meatus and the foramen for the 6th cranial nerve appeared perfectly normal. The dura mater lining the posterior fossa was entirely normal. However, within the jugular foramen was a granular pinkish-gray mass which did not extend quite above the level of the foramen into the posterior fossa (Fig. 1). With a saw the bone was divided just through the posterior margin of the jugular foramen. Thus laid open, we found a mass of pinkish-gray tissue involving all of the nerves of the jugular foramen, that is, the 9th, 10th, and 11th, in one mass, and attached to the posterior wall of the internal jugular vein. When this mass was removed and the jugular vein incised, it was found that a nodule of the tumor, measuring approximately 3 or 4 mm. in diameter, protruded into the vein (Fig. 2). The mass of tumor measured $2 \times 1 \times 1$ cm. The tumor did not have a sharp capsule and seemed

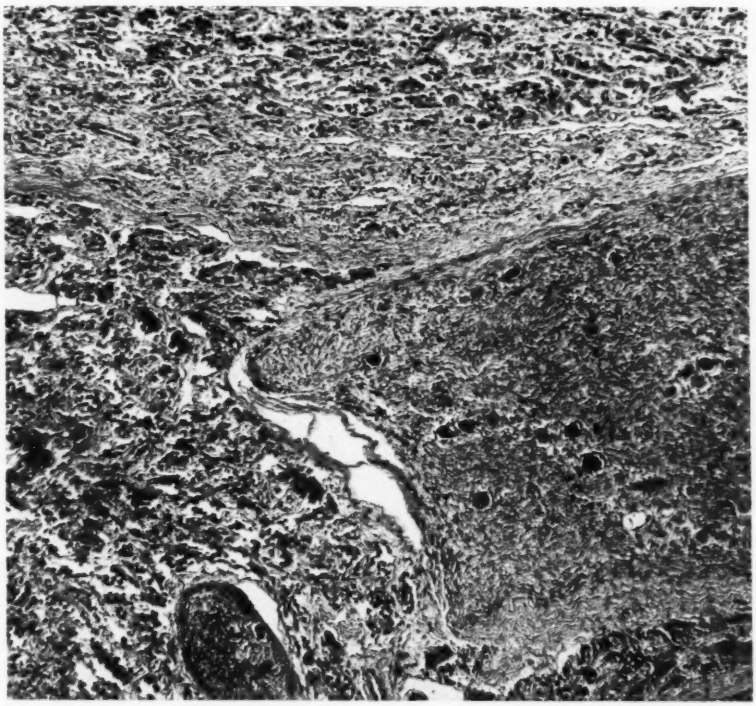

FIG. 3. Section through the vagus nerve which contains cells of the ganglion nodosum and is surrounded by the paraganglioma. (Hematoxylin and eosin ×70)

to involve all of the structures of the jugular foramen equally. The entire mass, including the wall of the jugular vein and the three nerves, was removed *en bloc*. The petrous pyramid was sectioned anteroposteriorly and longitudinally through the internal auditory meatus without disclosing any additional abnormality. On opening the mastoid process there was no evidence of tumor or infection.

No abnormalities were noted in the region of the carotid or aortic bodies. There were a severe fatty infiltration of the walls of the right cardiac ventricle and an acute interstitial pneumonia. The immediate cause of death was believed to be vagal inhibition secondary to the trauma of the operation acting on a heart already the seat of severe fatty degeneration, with a clinically unrecognized interstitial pneumonia being a contributing factor.

Microscopic Examination (Dr. Otto Saphir). Sections revealed a conglomerate mass of tumor imbedding large nerves and filaments, which contained many nerve cells, ganglia, venous sinusoids and the wall of the jugular vein (Fig. 3). The tumor was poorly circumscribed and was composed of loosely packed polygonal cells loosely arranged in small islands, nests and cords of varying sizes (Fig. 4). The cytoplasm was eosinophilic and contained numerous fine amphophilic granules (Fig. 5). The nuclei were moderately pleomorphic and varied considerably in size. They were round to oval and appeared to be of two types, one finely stippled with chromatin and the other containing coarse chromatin clumps. The stroma was composed of richly vascularized fibrous tissue which formed septa which surrounded the islands of tumor but failed to penetrate between the cells. In some areas, the septa became quite thick and col-

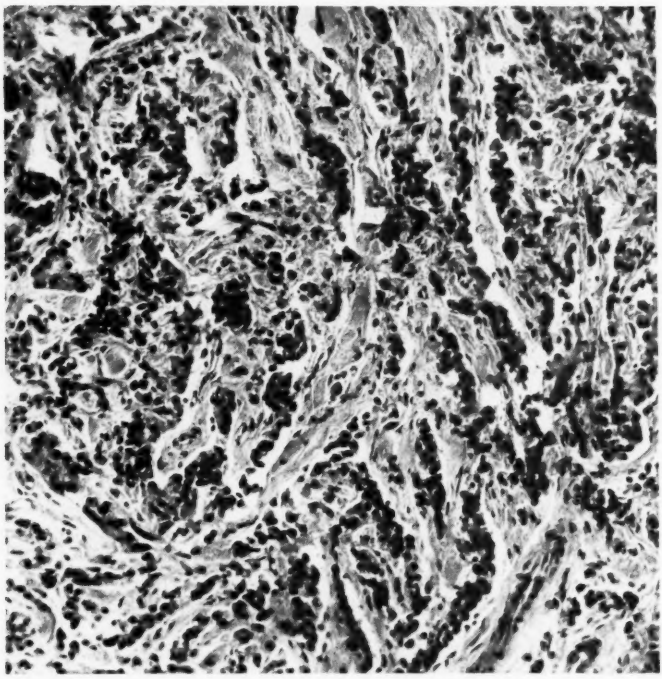

FIG. 4. Typical appearance of paraganglioma, with tumor cells arranged in cords. (Hematoxylin and eosin ×150)

lagenous. The ganglia and nerves were compressed and their epineurium was invaded by the tumor, but the structures themselves were free of tumor. Silver stains showed fragmentation and disappearance of axons. The tumor infiltrated the adventitia and media of the jugular vein, penetrating the intima in one area to form a pedunculated intraluminal tumor mass which appeared to be covered by endothelium. A similar pedunculated tumor was seen inside a vein within the tumor mass. There were also several cavernous spaces within the tumor which were lined by little more than a layer of endothelium (Fig. 6). Iron stains failed to reveal the presence of iron within the tumor and did not stain the cytoplasmic granules.

DISCUSSION

Having the typical appearance of a carotid-body tumor and not being metastatic, it seemed likely that this tumor arose from a structure similar to the carotid body, in this case the paraganglion intravagale of the ganglion nodosum.

There are only 4 other similar tumors in such a location mentioned in the literature. A comparative study of these cases shows that most of the signs and symptoms were indicative of involvement of the structures of the jugular foramen; this is particularly clear in the case of Dr. Semmes and in our case. In Stout's case (Lattes' Case 1) the neurological symptoms were mentioned only postoperatively, and they referred particularly to the 9th, 10th, 11th and 12th cranial nerves. In Lattes' Case 2, with a palpable mass in the neck, there was evidence of involvement

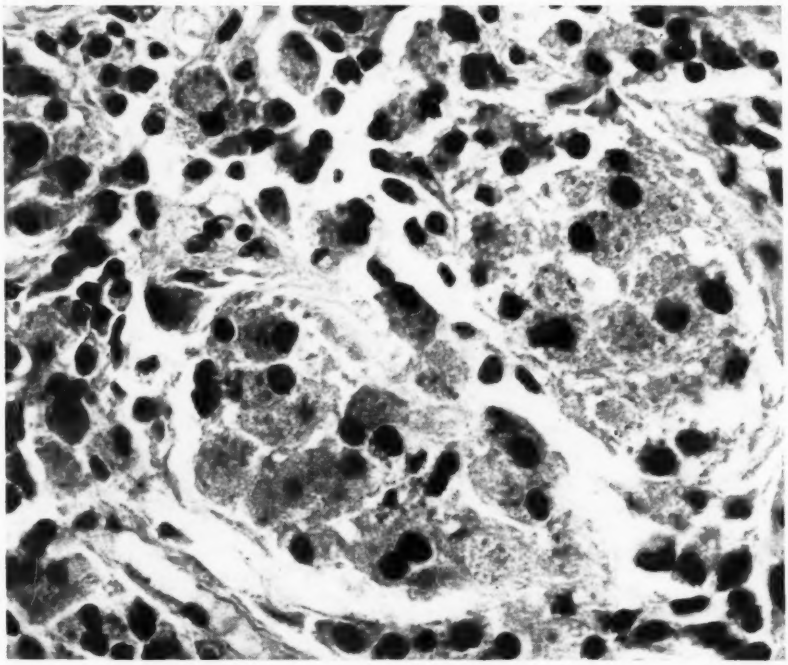

FIG. 5. Clusters of large polygonal cells, containing numerous fine amphophilic granules. (Hematoxylin and eosin ×570)

of the 11th and 12th cranial nerves. In his Case 4 no neurological findings are recorded. (His Case 3 was a tumor of the aortic body.)

In addition, pain in the back of the head and/or behind the ear on the affected side was present in at least 2 of the 5 cases. A palpable mass in the pharynx or in the side of the neck was recorded in 3 of the 5 cases.

Roentgenograms of the skull were negative in all but 1 case in which there was questionable enlargement of the jugular foramen.

Some of these tumors may be completely removed, sacrificing the 9th, 10th and 11th cranial nerves. In Stout's case an extracranial approach sufficed for complete removal. In our case the location of the tumor within the jugular foramen made surgical treatment very difficult, particularly as the tumor could not be seen at the operation. Had the tumor been exposed by resecting the floor of the posterior fossa lateral to the jugular foramen it would have been necessary to sacrifice the 9th, 10th and 11th cranial nerves and the internal jugular vein in order to remove the tumor. This, of course, would have been a difficult and hazardous procedure and the loss of these three important cranial nerves, particularly the entire right vagus, would have seriously handicapped the patient. Nevertheless there would seem to be no alternative and should we be presented with this problem again we would feel justified in carrying out such radical surgery as a life-saving measure.

All of these tumors, like nonchromaffin paragangliomas in other locations, were

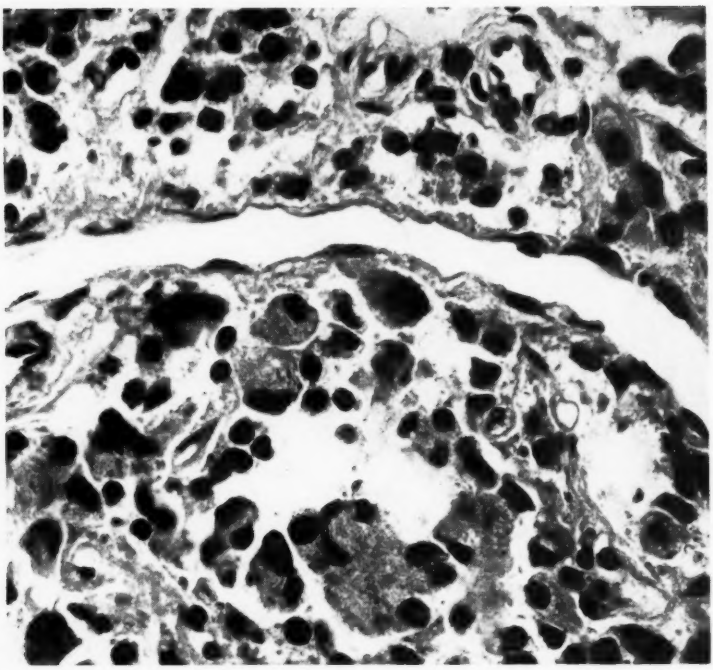

FIG. 6. Numerous cavernous channels formed by a single layer of endothelium course among the tumor cells. (Hematoxylin and eosin ×570)

quite vascular at operation with 1 exception (Stout's case). These tumors do not seem to respond to X-ray therapy. They apparently grow slowly (1 patient had an 8-year history) and may attain considerable size. On the other hand, some of them, though very small in size, may damage the 9th and 10th nerves so severely as to render the patient helpless with continuous vomiting.

SUMMARY

A case of nonchromaffin paraganglioma of the paraganglion intravagale of the ganglion nodosum is reported. It produced the typical clinical syndrome of the jugular foramen and continuous vomiting. A review of the literature disclosed that only 4 similar cases have been reported. All of these tumors are similar, histologically, to the tumors of the carotid body. It is believed that they have arisen from the paraganglion intravagale of the ganglion nodosum of the vagus nerve. Surgical treatment is difficult and hazardous and complete removal requires that the 9th, 10th and 11th cranial nerves and the internal jugular vein be sacrificed.

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TUMOR OF THE GLOMUS JUGULARE

FOLLOW-UP STUDY TWO YEARS AFTER ROENTGEN THERAPY

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In June 1950 a 35-year-old negro woman was discovered, after some 3 years of symptoms, to have a glomus jugulare tumor involving the left petrous pyramid and presenting through the left ear drum. A report of this case was made previously by Alexander, Beamer and Williams.[1] The patient had become almost completely bedridden. A carotid angiogram at that time delineated a large, vascular tumor of the left posterior fossa extending across the midline. Biopsy of the lesion through the external auditory meatus led to the diagnosis of a glomus jugulare tumor. This was considered inoperable at the time.

Subsequently the patient was given roentgen therapy, 2080 roentgen units (in air), through each of two 10 by 10 cm. occipital portals, administered over a 19-day period from July 6 to July 26, 1950. Six weeks later, from Aug. 29 through Sept. 29, 1950, she received, elsewhere, 9000 roentgen units over a right occipital portal.

FOLLOW-UP STUDY

The patient slowly but definitely improved. A graphic presentation of her neurological findings at the time of admission on June 24, 1950, before roentgen therapy, compared with the findings on July 10, 1952, 2 years after roentgen therapy, is shown in Fig. 1.

Briefly on *June 24, 1950*, before roentgen therapy, the findings were:

Subjective

Left facial weakness
Tinnitus and deafness of the left ear
Severe hiccoughs
Difficulty in deglutition and phonation
Ataxia
Headache

Objective

An audible bruit over the left mastoid
An almost complete left facial paralysis of the peripheral type
Complete deafness of the left ear
A tumor presenting in the left ear drum
Deviation of the palate to the right on phonation
Voice changes indicating a paralysis of her vocal cords
Paralysis of the left sternocleidomastoid muscle
Paralysis of the left side of the tongue
Ataxia of the left arm and hyperactive reflexes on the left side

On *July 10, 1952*, 2 years after roentgen therapy, the findings were:

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Subjective

No headache
No hiccoughs
Deafness of the left ear
Mild tinnitus of the left ear
No difficulty with deglutition and phonation
No ataxia

Objective

No bruit over the head
A smaller tumor presenting in the ear than before roentgen therapy
Only slight evidence of a residual facial palsy on the left
Deafness of the left ear
No palatal paralysis
Normal voice
Paralysis of the left sternocleidomastoid muscle, complete
Paralysis of the left tongue, complete
No ataxia
Normal deep tendon and superficial reflexes

In summary, there has been very marked subjective improvement in headache, ataxia and hiccough. The patient has been able to do her own housework. There has been objective evidence of shrinkage of the intracranial mass and disappearance of the bruit over the left mastoid, almost complete disappearance of the left facial paralysis, disappearance of the 9th and 10th cranial nerve palsy and loss of spasticity of the entire left side of the body.

	June 24, 1950 Before Roentgen Therapy	July 10, 1952 After Roentgen Therapy (2 yrs.)
Audible Bruit over Skull	++	0
Facial Paralysis Left	+++	±
Tumor Left Ear	++++	+++
Deafness Left	++++	++++
Palatal Paralysis	++++	0
Vocal Cord Paralysis	+++	±
Paralysis Sterno-cleido mastoid left	++++	++++
Paralysis Tongue Left	++++	++++
Ataxia	+++	0
Headache	+++	0
Hiccoughs	++++	0

FIG. 1. Graphic demonstration of improvement shown by patient following roentgen therapy.

In addition to the above data, carotid angiography was performed on the last day of her first course of roentgen therapy, July 26, 1950, and again on May 26, 1951, approximately 8 months after her last course of roentgen therapy. Although one cannot draw far-reaching conclusions from the failure of an intracranial mass lesion to fill with diodrast at the time of angiography, there seemed to be less vascularity of the tumor after roentgen therapy and the mass appeared smaller (Fig. 2). Whether this was because of a variation in the films it is impossible to state. The two angiograms were done by the same operator, using the same roentgenographic equipment. The absence of the intracranial bruit at the time of the second procedure is confirmatory evidence of the decreased vascularity of the tumor.

DISCUSSION

Of the reports in the medical literature concerning the effects of roentgen therapy on carotid body-like tumors, only those of Bevan and McCarthy[2] and Lahey[3] expressed any optimism whatever. In the patient reported by Bevan and McCarthy[2] there was striking diminution in the size of the tumor in the neck 13 months after roentgen therapy. In 3 patients described by Lahey,[3] definite recession in the size of the tumors in the neck occurred after roentgen therapy. All other reports regarding these tumors have been pessimistic about any benefit that might be received from roentgen therapy.[4]

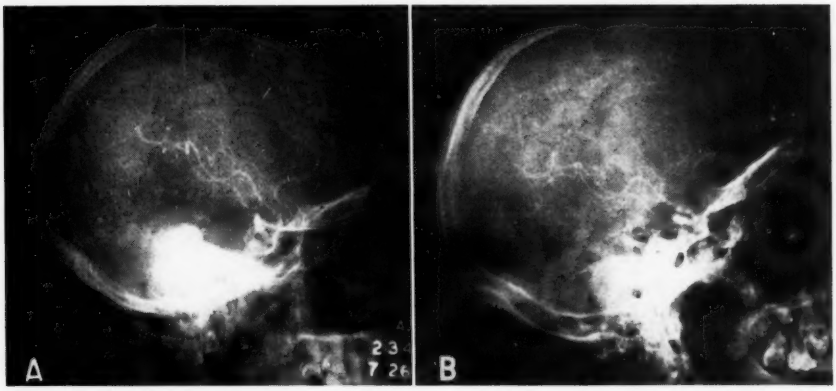

FIG. 2. Left carotid angiograms. (A) Before effect of roentgen therapy. (B) 10 months after roentgen therapy. The tumor mass in the petrous region does not fill with the same concentration of diodrast as in (A).

It suffices to say that if a tumor of the glomus jugulare is amenable to surgical removal, this should be done. However, in cases such as the one presented herein, in which there has been obvious central nervous system involvement and invasion of the bone of the base of the skull, the hazard of an attempt at surgical removal is greater than can be justified. The course of patients with tumors of the glomus jugulare is known to be long, sometimes years, before the individual comes to operation, but there has not been evidence of spontaneous recovery without surgery in previous cases. Except for the fact that our patient stated that at one time she had had numbness of the left side of her face and palate which had recovered by the time she was first examined, her course seems to have been steadily in the direction of increasing involvement of the central nervous system until she received roentgen therapy. Since then her course has been one of steady improvement from the neurological point of view.

Previous sporadic attempts at carotid angiography in cases of tumors of the glomus jugulare did not result in satisfactory filling of these tumors with diodrast. Perhaps the fact that the tumor in the present case filled so well and was of a more vascular nature than others, accounts for its apparent sensitivity to radiation therapy. There seem to be no atypical features in the microscopic structure of this tumor that might have led to a prediction as to its radiosensitivity.

There is little doubt, however, that the tumor in this individual did respond to roentgen therapy and in future cases in which surgery is not feasible, a thorough trial of roentgen therapy would be justifiable.

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LIPOMA OF THE SPINAL CORD ASSOCIATED WITH KLIPPEL-FEIL SYNDROME

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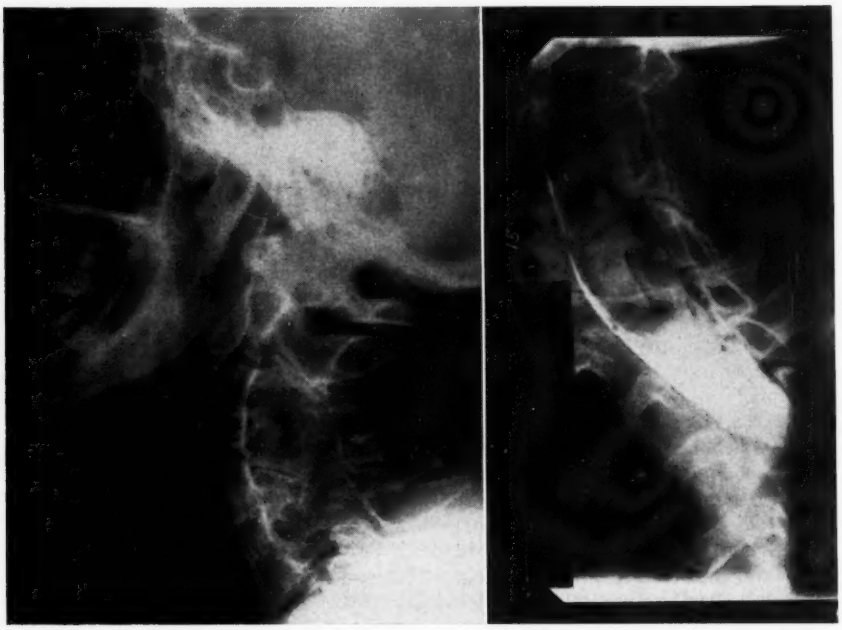
Central nervous system manifestations associated with the Klippel-Feil syndrome are not uncommon, and are probably caused by a congenital myelodysplasia. This naturally raises the question as to whether in some cases the Klippel-Feil syndrome may be associated with an intramedullary tumor, particularly since the central nervous system symptomatology resembles that of syringomyelia in many instances. It is also well recognized that the symptomatology of syringomyelia and intramedullary tumor may be indistinguishable. An intramedullary tumor (ependymoblastoma) was found at autopsy in a patient of Turnbull's,[6] but his case was not one of Klippel-Feil deformity. Du Toit[2] considered the probability of an intramedullary tumor in his patient with Sprengel's deformity. The possibility of an intramedullary tumor was also mentioned in the case reported by Avery and Rentfro.[1] In Nery's[5] case the Klippel-Feil syndrome was most likely associated with an intramedullary tumor. A myelogram disclosed a block at the level of the 7th thoracic vertebra. His patient died rather suddenly from respiratory embarrassment. Unfortunately, a postmortem examination was not obtained. Proven intramedullary tumors, however, have not been reported heretofore, as far as the author is aware, and the following case is therefore presented.

CASE REPORT

A 48-year-old female, referred by Dr. Harry Subin of Atlantic City, N.J., was admitted to Temple University Hospital on Mar. 11, 1949. She had had difficulty in walking and in raising both arms as long as she could remember. The right extremities were more involved than the left. She could never raise her hands above the level of her shoulders but was able to comb her hair with the left hand. During the last year she had experienced progressive difficulty in walking and she complained of pain in both forearms, especially the right. She described a "numb feeling" in both forearms. The numbness appeared to be greater on the left side and prevented her from being able to mend her clothes. She denied any bowel or bladder difficulty.

Examination. She had large, heavy-set features. Her neck was shortened, and the hair line extended to the mid-cervical area. Below the hair line there was a huge pad of fat which extended from C7 to T4 and gave the appearance of a gibbous deformity (Fig. 1). The gait was spastic so that she barely shuffled along. The right lower extremity was more spastic than the left and it was more difficult to raise the right

Fig. 1. Photograph of patient showing low hair line, short neck and the pad of fat posteriorly.



FIGS. 2 and 3. (*Left*) Roentgenogram of cervical spine showing fusion of the cervical segments. Note widening of the cervical canal. (*Right*) Pantopaque myelogram delineating a block at the cervicothoracic junction.

limb. Spasticity involved all four extremities but the lower limbs were more severely affected. The left arm could be raised to the level of the head but the right arm could be elevated only as high as the shoulder. Deep tendon reflexes were hyperactive bilaterally. A Hoffmann sign was present on both sides. Abdominal reflexes were not elicited. The Babinski sign could not be demonstrated on either side. Sensation to pain and temperature was decreased bilaterally from C3 to T5. Touch sensation was decreased over the same areas but particularly over the 4th thoracic dermatome. Position and vibration sensations were decreased in both upper limbs but more so in both lower limbs. There was marked dyskinesia in the right upper limb and dysmetria in both upper limbs.

Spinal puncture revealed a complete manometric block on Queckenstedt maneuver. The only spinal fluid abnormality was a protein of 82 mg. per cent. Roentgenograms showed an anomalous fusion with a deformity in the cervical area. The 3rd, 4th and 5th cervical segments were fused together, and the 6th and 7th segments were likewise fused. Apparently seven cervical segments were present. In addition to the fusion, the involved vertebral bodies were relatively narrower in their anteroposterior dimension. The vertebral canal was quite large. The first two cervical segments appeared normal (Fig. 2). Pantopaque myelography (Fig. 3) was carried out to delineate the extent of the lesion, if possible, and to perhaps determine whether it was intra- or extramedullary. The pantopaque injected into the lumbar region was brought to the cervical region without difficulty. An unusually large vertebral canal was demonstrated. The morphology at the level of the foramen magnum was not well delineated and some of the pantopaque entered the skull. The caliber of the cervical vertebral canal was well shown; the canal measured $3\frac{1}{2}$ cm. in transverse dimension and 3 cm. in the anteroposterior dimension. In the supine position it was not possible to bring the pantopaque any higher

than the level of the 7th cervical vertebra even though the table was tilted to an angle of about 60° with the horizontal.

Operation. In a sitting position it was difficult to flex the chin forward and this made exposure difficult. Muscles and fascia were divided and lamina C1 to C7 were removed. The canal was quite widened and the dura mater was tense. The dura was opened in the midline and there immediately protruded a large, rounded, yellowish mass, approximately the size of a fountain pen. It was intramedullary and occupied almost the entire substance of the cord from C7 to the foramen magnum. A portion of the tumor was dissected free at its lower aspect, but as the dissection was carried upward, it was obvious that the tumor was intramedullary and had made a shell of the cord around itself. In the upper part, at the level of C2,

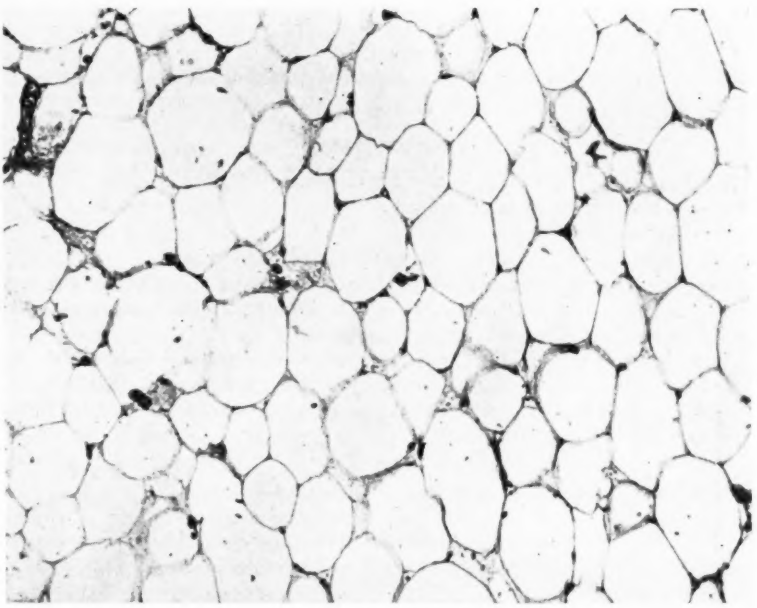


FIG. 4. Photomicrograph of the tumor, which is a typical lipoma.

the tumor was split in the midline and the incision was carried to the ventral aspect of the dura mater. No cord substance was encountered through the incision, indicating that practically all of the spinal cord had been replaced by fatty tumor and that only remnants of the lateral columns remained on either side. The dura mater was left open so as to obtain full decompression. Part of the tumor was removed for biopsy study. Muscles and fascia were closed with stainless steel wire. Skin was closed with interrupted stainless steel wire. Time of operation—2½ hours.

Histological Study. The appearance of the tumor was typical of a lipoma (Fig. 4).

Course. Convalescence was uneventful. There was a decided improvement in her walking with a definite return of motor power in all four extremities. She had residual pain particularly in the right forearm. Her neurological findings, however, remained unchanged.

She has been seen at periodic intervals, and when last examined in April 1953 (4 years later) she was still able to ambulate under her own power and her neurological picture appeared to have reached a stationary level.

DISCUSSION

It would appear that the Klippel-Feil syndrome is an expression of a dual congenital anomaly involving both the skeletal and the neurogenic tissues. The central nervous system manifestations are usually indistinguishable from those of syringomyelia and are probably caused by a spinal dysraphism. This being so, it is not unlikely that embryonal cell rests may give rise to tumor formation. Du Toit[2] stated that in his case all the symptoms pointed to a syringomyelia but the possibility of an intramedullary tumor had to be considered. In Nery's[5] case the syndrome was undoubtedly associated with a tumor but unfortunately autopsy findings were unavailable. In the case presented herein the intramedullary lipoma might have arisen from misplaced cell rests accompanying a myelodysraphia. It is obvious that more operative or autopsied material is needed in these cases. In those cases in which symptoms of syringomyelia are present, the cause may well be an intramedullary tumor.

COMMENT

Attention should be called to the widened cervical canal, which has been reported by others.[3,4,5] Such a widening could be caused by the presence of tumor formation although it is most likely the result of the congenital deformity with lack of fusion of bilateral osseous centers of the cervical spine.

Some patients with Klippel-Feil syndrome show a pad of fat over the cervicothoracic junction, as in the author's case. Whether or not this has any relationship to the intramedullary lipoma is only speculative.

It is rather remarkable that such a paucity of symptoms exists with such a massive lesion which nearly completely destroys the spinal cord so that only a mere shell remains. The author's patient, furthermore, appeared to improve following surgery and continues to remain in a relatively stationary neurological status, 4 years following operation. The slow growth of the tumor and the benefit of decompression may explain this in part.

SUMMARY

A case is presented of Klippel-Feil syndrome associated with an intramedullary lipoma of the spinal cord. Operative intervention has apparently arrested the progression of the disease. Others cases of Klippel-Feil syndrome associated with tumor undoubtedly exist but more pathological material is essential to prove this point.

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ESOPHAGORESPIRATORY COMPLICATIONS OF TREATMENT OF NEUROSURGICAL PATIENTS

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This paper is a report of 2 neurosurgical cases in which the clinical features of esophagorespiratory fistula developed postoperatively during a period when a Levine tube and tracheal cannula were concomitantly in use. We have failed to find reports of this complication in the literature up through November, 1952.

Case 1. #52-5366. G.A.G., a 22-year-old white female, in September, 1949 had headaches, occasional nausea, vomiting, and intermittent diplopia without lethargy or signs of meningitis. She recovered after several weeks and remained well for over 2 months. In January, 1950 hyperkinesia first appeared in the head and hands. This increased in severity so that by late Summer, 1951 she exhibited extensive, non-patterned, involuntary movements of all members of the body, defective balance, ataxia, and dysarthric speech.

She was admitted to the University Hospital on May 19, 1952.

1st Operation. A mesencephalic pedunculotomy of the lateral two-fifths and medial two-fifths of the right crus was performed under endotracheal general anesthesia.

Course. A transnasal Levine tube through which fluids, food and anticonvulsant medication were passed was in place from the 2nd to the 9th postoperative day. She made an uneventful recovery and was discharged 15 days after operation.

She was readmitted on Sept. 27, 1952.

2nd Operation. On Oct. 1, 1952 a similar mesencephalic pedunculotomy was performed on the left side.

Course. A Levine tube was passed on the 3rd postoperative day. On the 8th day signs of respiratory obstruction developed. Roentgenogram of the chest taken with a portable machine revealed no evidence of atelectasis or pneumonia. However, the patient continued to manifest signs of respiratory difficulty. A tracheotomy was therefore carried out on the same day. From the 14th to the 18th day tarry stools were passed. On the 16th day some bloody material was aspirated through the Levine tube. Transfusions of 500 cc. whole blood were given on the 16th and 17th days. After the 18th day no signs of gastrointestinal bleeding were observed. On the 21st day the patient coughed some formula through the tracheotomy tube. The Levine tube was promptly removed and fluid intake was maintained by hypodermoclysis for the next few days.

An otolaryngologic consultation was obtained on the 24th day. A swallow of water containing methylene blue was coughed out of the tracheotomy tube after 10 seconds. This was considered presumptive evidence of an esophagorespiratory fistula. Several unsuccessful attempts at bronchoscopy and esophagoscopy were made. On the 33rd day these procedures were successfully carried out under general anesthesia. At 35 cm. from the upper teeth an erosion was demonstrated on the posterior and lateral esophageal wall. Here the mucosa bled easily. On bronchoscopy, "a redness but no fistula or erosion" was noted at the level of the carina. At this time a swallow of lipiodol failed to reveal evidence of a fistula. The tracheotomy tube was removed on the same day and a Levine tube was reinserted. By the 41st day the patient took very small amounts of food by mouth without difficulty. The Levine tube was then removed. By the time of her discharge from the hospital on the 45th day she was taking a regular diet.

Case 2. #52-892. J.D.L., a white man aged 56 years, was transferred to the Neurosurgical Service in a comatose condition. Pneumoencephalography had revealed evidence of a mass in the right parietal region.

Operation. A right postcentral craniotomy was carried out on Jan. 27, 1952 and a meningioma measuring $8 \times 8 \times 1\frac{1}{2}$ cm. was removed under endotracheal general anesthesia.

Course. On the 3rd postoperative day a Levine tube was passed. On the 6th day the patient began to show signs of respiratory obstruction secondary to hypopharyngeal relaxation. At first this could be readily relieved by altering his posture, but by the 8th day it was deemed necessary to perform a tracheotomy.

The Levine tube was changed from one side of the nose to the other about every 7th day. Frequently, several attempts were necessary in order to pass the tube. On the 18th day the tube feedings were supplemented by small amounts of oral fluids. On the 25th day the patient coughed violently each time he attempted to swallow liquids. Roentgenogram of the chest showed signs of bilateral pneumonitis. All oral feedings were discontinued.

An otolaryngologic consultation was obtained on the 27th day, and the tracheotomy tube was removed. The opinion was expressed that an esophagorespiratory fistula had developed "secondary to friction from both the Levine tube and tracheotomy tube." It was also thought that irritation from the Levine tube and consequent edema of the mouth of the esophagus and arytenoids with spillover into the trachea might be responsible for the signs exhibited by the patient. Indirect laryngoscopy on the same day revealed moderate edema of the epiglottis, arytenoids, and true and false vocal cords. The Levine tube was removed and fluid intake was maintained by hypodermoclysis. On the 31st day, oral liquids in small amounts were tolerated without difficulty. Thereafter, the diet was progressively advanced until the patient was discharged on the 42nd postoperative day, able to take a full diet.

DISCUSSION

The majority of cases of esophagorespiratory fistula recorded in the literature are ascribable to congenital atresia and carcinoma of the esophagus. A less frequent causative factor is aortic aneurysm.[2] In 1950 Coleman[1] reviewed the literature on nonmalignant acquired esophagotracheobronchial fistula reported during the previous 32 years. Of 75 cases, 21 were caused by the swallowing of foreign bodies, instrumentation, and other "trauma." The remainder were imputed to esophageal diverticulum and infectious agents, including syphilis. In 16 instances the etiology was undetermined. Lukens and Ono[5] alluded to 2 cases of esophagorespiratory fistula secondary to the pressure of tracheal cannulae described by Sirot in his thesis of 1898.

The use of tube feedings to insure adequate nutrition for the comatose or recalcitrant patient is a familiar and highly valuable procedure. The use of a tracheotomy tube in the patient with respiratory obstruction or otherwise threatened with hypoxia is likewise familiar and frequently a life-saving measure.[6] The use of either tube alone is not without danger. Esophagitis has been described[3,8] following the use of Levine tubes, and standard texts in otolaryngology[4,7] warn of the dangers of necrosis and stricture of the trachea caused by poorly placed and improperly selected tracheotomy cannulae. The combination of Levine and tracheotomy tubes is less frequently necessary than either tube alone, but its use has been growing, especially in the field of neurosurgery. The complication herein reported is rare in our experience and in no sense suggests that the combined use of the measures referred to should be abandoned. We wish merely to point out the potential dangers inherent in the procedure. Marked coughing and/or the appearance of food particles in the tracheotomy tube during the act of deglutition strongly suggests but does not conclusively establish the existence of an esophagorespiratory fistula. Incompetence of the deglutitory mechanism may on occasion be associated with edema of the gullet and/or laryngeal apparatus. The only certain criteria of

the diagnosis of esophagorespiratory fistula are (a) radiographic demonstration and (b) direct visualization by bronchoscopy and esophagoscopy. Measured in terms of these criteria it cannot be conclusively asserted that our patients suffered from esophagorespiratory fistula. The diagnosis in each case was reached by inference. However, irrespective of the existence or nonexistence of such fistulae, the complications presented above in connection with combined use of Levine and tracheotomy tubes were characterized by aspiration of food into the lower respiratory tract. They carry every potential for morbidity and mortality that attaches to the existence of esophagorespiratory fistula.

In view of the potential pressure necrosis occurring along the apposed surfaces of the lower respiratory tract and esophagus in consequence of the presence of two mechanical agents (tracheal cannula and Levine tube), the following recommendations seem warranted: (1) Awareness and unerring vigilance on the part of the clinician and nursing staff of the possibility of the complication here described and its clinical signs. (2) Replacement of the Levine tube by a tube of smaller caliber. (3) Care in the selection and placement of the tracheal cannula. In order to reduce the bulk of the intraesophageal mass, we have recently adopted a transnasal polyethylene tubing (external diameter 2.5 mm.) in place of the Levine tube.

SUMMARY

1. Two cases of esophagorespiratory complications which developed during the concomitant use of Levine and tracheotomy tubes are recorded.

2. A review of the literature failed to demonstrate previous similar reports.

3. The use of transnasal polyethylene tubing in place of the standard Levine tube is recommended.

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POSTTRAUMATIC OCULOCARDIAC SYNDROME FROM A NEUROSURGICAL POINT OF VIEW

REPORT OF A CASE

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In cases of head injury the differential diagnosis of an intracranial hematoma is important. A useful diagnostic criterion is the slowing down of the pulse rate caused by increased intracranial pressure; this sign in a case of head injury is considered almost pathognomonic of an intracranial hematoma.

However, trauma of the eye or orbital region may cause a retardation of the heart rate without any existing intracranial hematoma. This phenomenon is produced by the oculocardiac reflex, which is well known therapeutically in cases of paroxysmal tachycardia, in which pressure on the eyeball may abort the attack.

In the present paper a case is reported in which a marked oculocardiac reflex followed a blow to the eye.

CASE REPORT

J.Nr. 620/1946. B.P., a 25-year-old man, was admitted with a diagnosis of intracranial hematoma.

About 4 hours previously he had been involved in a fight and received a blow to his left eye. He tumbled down, bumped his occiput against the pavement and fainted. After some minutes of unconsciousness he recovered, and was taken to the out-patient department of another hospital. On examination there, about ½ hour after the accident, he was slightly confused but well oriented. On the left side a small subconjunctival hematoma and a slight mydriasis were found. The left pupil reacted sluggishly to light. There were no other signs of injury. Roentgenograms of the skull did not disclose any fracture.

The pulse rate was 60 per min., and the B.P. was 130/70. Shortly afterwards the pulse rate dropped to 44, and during the next 2 hours it became progressively slower until it reached 38 per min. The B.P. was unchanged and the patient was fully conscious and mentally unaffected.

The left subconjunctival hematoma and the palpebral edema had increased, and the left pupil was more dilated. The clinical picture gave suspicion of an intracranial hematoma and the patient was transferred to our clinic.

Examination, 4 hours after the accident. The patient showed no signs of slow cerebration. He was mentally unaltered and without any retrograde amnesia.

The pulse rate was 36 per min. B.P. was 120/80. Respiratory rate was 20 per min.

The left "black eye" showed considerable palpebral edema, and a subconjunctival hematoma was found. The movements of the eyeball were impaired, with a partial paresis of the rectus inferior and oblique muscles. The left pupil showed mydriasis with diminished reaction to light. The vision was 1.5 bilaterally. The media were clear and the left fundus showed a slight retinal edema. The intraocular pressure measured 12 mm. in both eyes; it was thus within normal limits, but on the threshold of a bulbar hypotonia.

Course. The clinical picture was ascribed to an exaggerated oculocardiac reflex elicited by the intraorbital hematoma and edema, as a thorough neurological examination had not revealed any other signs of an intracranial hematoma. His course was uneventful. On the day after admission the pulse rate increased to above 40 and the patient did very well. On the 4th day he left the hospital on his own volition. He still had a slight left-sided subconjunctival hematoma and a slow pulse rate.

* Director: Professor Herbert Olivecrona.

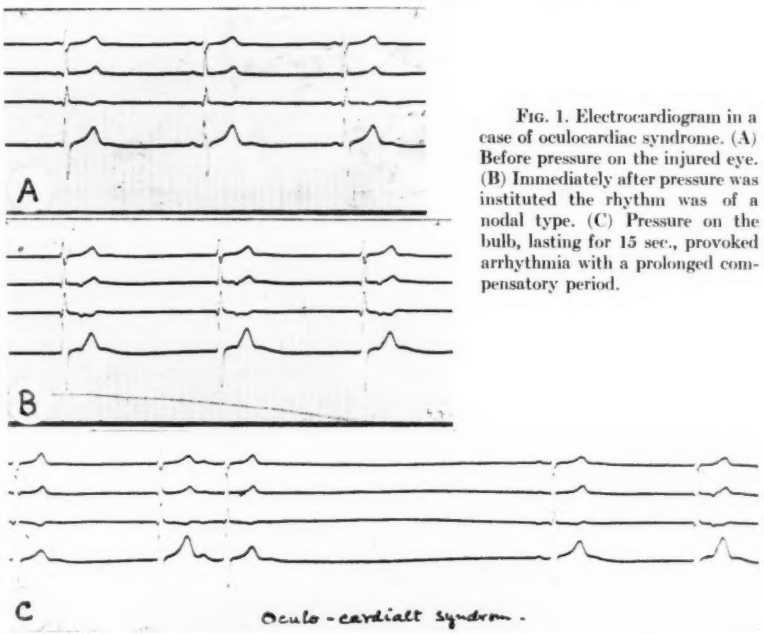

FIG. 1. Electrocardiogram in a case of oculocardiac syndrome. (A) Before pressure on the injured eye. (B) Immediately after pressure was instituted the rhythm was of a nodal type. (C) Pressure on the bulb, lasting for 15 sec., provoked arrhythmia with a prolonged compensatory period.

Electrocardiographic Examination. On the 2nd day after admission electrocardiography was performed. The tracings showed a bradycardia with a rate of about 40 per min., but were otherwise normal (Fig. 1 A).

After closing the eyelids digital pressure was exerted upon the eyeballs.

Pressure on the left, injured eye provoked an extreme bradycardia. After a latent period of 15 sec. an arrhythmia ensued, with considerable lengthening of the compensatory period. After a heart beat of 0.7 sec. there was a pause of 3.6 sec. It may be presumed that this prolongated compensatory period was the beginning of an asystolia. The electrocardiogram revealed that the pressure against the orbital content also provoked a nodal rhythm (Fig. 1 B), which disappeared immediately on relieving the pressure.

On the other hand, the same or even a stronger pressure exerted against the right healthy eye could not provoke more than a slight slowing down of the pulse rate, without any other pathological findings on the electrocardiogram. Of course, too strong a pressure was not exerted, as there might have been a risk of retinal damage.

The trauma caused by the blow thus seemed to have sensitized the left eye, eliciting a spontaneous oculocardiac reflex.

Control Examination. The patient was again examined 1½ years after the accident. Neurological findings and both eyes were normal. The pulse rate at rest was slightly above 60 per min. Pressure on either eyeball caused a slowing down of the pulse to a rate of 50 per min. The electrocardiogram did not reveal any pathologic changes of the kind observed at the acute stage of the accident.

DISCUSSION

The oculocardiac reflex is often called the "Aschner-Dagnini reflex" as these authors,[1,7] in 1908, independently described a reflex that was elicited by pressure on the eyeball and provoked a slowing down of the pulse rate.

Detailed accounts of a more general nature are available in a number of publications, i.e. by Barré and Crusem,[5] Daniélopolu,[8] Van Dooren,[19] Laignel-Lavastine,[10] Petzetakis,[13] and Söderbergh.[17]

The centripetal pathway from the sensory innervation of the orbit is considered to take place through the trigeminal nerve to the pons, where the center of the reflex arc is situated. The efferent fibres pass through the vagus to the heart.

The oculocardiac reflex may be elicited by digital compression of the eyeball. However, irritation of the orbit, even after removal of the bulb, will elicit the reflex. Retroganglionic section of the trigeminal sensory root abolishes the reflex.[1,16]

Special apparatuses for more quantitative evaluation of the reflex, e.g. the oculocompressor of Barré,[4] have been invented. At a threshold pressure of about 500 gm. the reflex is considered positive if the pulse is slowed more than 12 beats per min.[5] The latent period in eliciting the reflex was found to range between $1\frac{1}{2}$ to 25 sec.[5]

The reaction is proportional to the degree of pressure employed. By extreme pressure a pause of several seconds may ensue between the cardiac contractions, as has been demonstrated on electrocardiographic tracings by several authors. Daniélopolu[8] and Petzetakis[13] found a duration of 5 to 6 sec. between the QRS-complexes, thus a pulse rate of about 10 to 12 per min. If too great a pressure is exerted the heart may come to a standstill in diastole. The cardiac rate returns to normal in the majority of cases within 20 sec. of the release of pressure.[9]

As a clinical test, the oculocardiac reflex has been used, especially by French investigators, who found it abolished or weakened in cases of pseudobulbar palsy,[6] tabes and general paralysis.[15] The oculocardiac reflex is suppressed by atropine.[13]

Aschner[1] stated that the reflex was accompanied by increased intracranial pressure; Rebattu and Josserand[14] found a parallelism between the retardation of the pulse rate and the increase of intracranial pressure.

Trauma of the Eye and Oculocardiac Reflex. Aubineau[2] experimentally produced a prolonged oculocardiac reflex by means of *subconjunctival* injections of saline solution. In 2 cases he obtained a slowing down of the heart rate to 34 and 36 pulsations per min. In cases of glaucoma with *periocular* edema similar findings have been published by Mérigot de Treigny.[11] A rise of the intra-ocular pressure, however, will not elicit the reflex. As shown by Aschner[1] in animal experiments, injections of solutions into the eye itself had no effect on the pulse rate.

In *orbital surgery* the reflex has been observed after enucleation of the eye with subsequent intraorbital hematoma. Bailey[3] reported an interesting case of an accentuated oculocardiac reflex elicited by the pressure of an intraorbital hematoma following enucleation. At the time of the operation, as the optic nerve was cut across, a severe hemorrhage occurred. The bleeding was controlled by sealing the operative conjunctival wound with a water-tight suture. The pent-up blood in the orbit caused a rise in the intraorbital pressure. As the patient was about to leave the operating room, he went into a state of apparent shock which was accompanied by an alarming slowing of the pulse. It became progressively slower until it reached 30 per min. Each pulse wave, however, was full and strong. This state of affairs continued for almost 2 hours before it suddenly dawned upon the surgeon that he was confronted with a very much exaggerated oculocardiac reflex.

Vialard[20] also described a case of spontaneous and inopportune oculocardiac reflex following enucleation of the eye after 6 hours. The pulse rate went down to 40 per min. and the patient was in a state of bradycardia and extreme fatigue for 3

days until the surgeon opened the compressing bandage over the orbit and removed a hematoma. Then the pulse rate returned to a normal of 75 beats per min.

Publications of a posttraumatic oculocardiac reflex are rare. Thus Mougeot and Duverger,[12] among 190 cases of war wounds of the orbital region, found only 5 cases of bradycardia, and none of them could with certainty be referred to as presenting an oculocardiac syndrome. Szokolik[18] reported 3 cases of bradycardia in patients with retrobulbar hematoma. In 1 of them, in which the hematoma was caused by a stab wound of the orbit, the pulse rate had slowed to 44. He discussed whether this state was ascribable to the intracranial hematoma or whether it was only a matter of "Aschner's symptom."

In cases of head injury the detection of an existing intracranial hematoma is important. With orbital lesions a prolonged and accentuated oculocardiac reflex may result in general vegetative symptoms, such as sweating, vertigo, faintness, substernal constriction, epigastric pain, nausea and vomiting, because of the elicitation of other oculovisceral reflexes.[8] In such cases the possibility of an oculocardiac syndrome should be kept in mind as a differential diagnosis.

The *treatment* instituted, if the symptoms do not disappear spontaneously, may be removal of an intraorbital hematoma and hemostasis by means of artery clips instead of tamponade of the orbit. Administration of atropine is said to suppress the reflex and may be of some value. The most rapid and efficacious method of controlling a prolonged oculocardiac reflex is considered by Mérigot de Treigny[11] to be a retrobulbar injection of novocain-adrenalin. He reported a case of intraorbital hematoma, following enucleation of the eye, in which the pulse rate dropped to 30 per min. After an intraorbital injection of novocain the pulse very soon returned to the normal rate of 60 per min.

SUMMARY

A case of oculocardiac syndrome following a blow to the left eye of a man aged 25 is reported. In a few hours after injury extreme bradycardia occurred with slowing down of the pulse rate to 36 per min. without any other symptoms of cerebral impairment.

Pressure on the bulb of the injured eye accentuated the bradycardia and almost provoked asystolia, which was recorded on the electrocardiogram.

The mechanism of the reflex and the differential diagnosis of an intracranial hematoma are discussed.

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INTRACEREBRAL CRYPTOCOCCIC GRANULOMA

CASE REPORT

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In their review of 178 cases of cryptococcosis with involvement of the central nervous system, Carton and Mount[3] found 42 cases in which operation had been performed. Of these patients, 9 were shown to have single or multiple granulomatous masses in the brain or spinal cord, either during operation or at autopsy.

Further perusal of the literature revealed only 5 cases in which a sizable cryptococcus granuloma was removed from the brain surgically. The first case was reported by Gáspár[6] in 1929. His patient had a 6 cm. mass in the left parietal region and died 3 weeks after operation. The second case was one of a cryptococcus granuloma in the left cerebellar hemisphere, reported by Dickmann, Veppo and Negri.[5] Their patient was discharged against advice 1 month after surgery with evidence of generalized infection. In the third case, reported by Swanson and Smith,[8] a granulomatous mass was completely removed from the right cerebellar hemisphere. The patient showed evidence of recurrence 4 months later and died 5 days after a second operation. In the fourth case, reported by Krainer, *et al.*,[7] there was a large cystic mass in the left "subfrontal" region. Their patient was living and well up to 11 months after operation, although he had evidence of systemic cryptococcosis. In the fifth case, reported by Daniel, Schiller and Vollum[4] there was a mass in the left cerebellar hemisphere which contained cryptococcic bodies in the paraffin sections. The spinal fluid showed normal findings, but the nasal secretions contained the yeast bodies. Their patient died 5 months after surgery.

The following report is the 6th recorded case of a discrete, sizable intracerebral cryptococcic granuloma which was successfully removed.

CASE REPORT

A white man, aged 35, had been in excellent health until July 9, 1952. On that day, while he was working on a construction job in the hot sun, he suddenly collapsed, became unconscious and had a series of convulsions, characterized by rigidity and rolling of the eyes and face to one side. He was immediately taken to a nearby hospital where he regained consciousness after a few hours. A spinal tap was done the next day. The CSF was reported to be clear and colorless, with a cell count of only 1 per c.mm., total protein of 33 mg. per cent and a type A/B

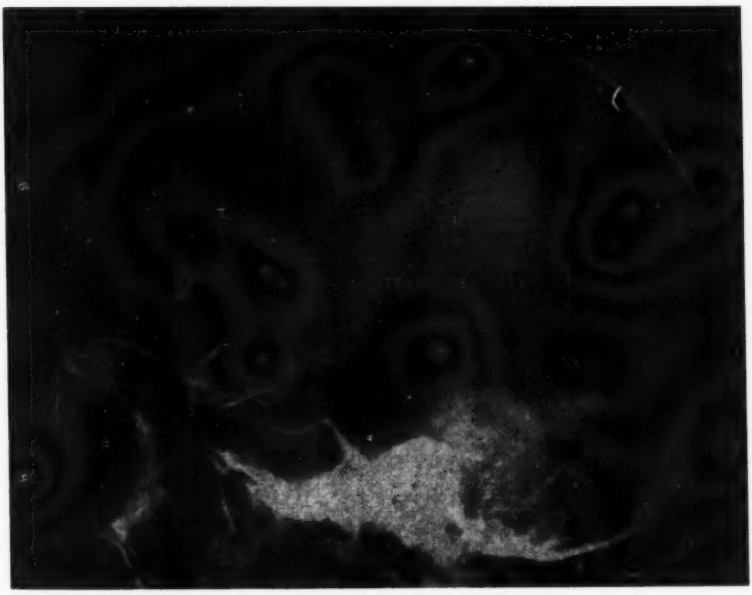

FIG. 1. Roentgenogram showing calcified masses in right frontal lobe.

colloidal gold curve. No culture was taken. The patient improved and was sent home on dilantin and phenobarbital medication. Roentgenograms of the skull were subsequently taken at another clinic and showed "calcified spots in the brain." The patient was admitted to this hospital for further diagnosis and treatment.

Examination. On admission the patient appeared slightly undernourished, but was not acutely ill. He had no complaints. He was worried over the possibility of another seizure. His past history was not remarkable. He had never been out of this country.

Positive neurological findings were a fine rapid nystagmus on lateral gaze to either side, absence of the left abdominal reflex, hyperactive left knee reflex and an extensor plantar response on the left. The fundi were normal. Roentgenograms of the skull showed 5 round "balls" of calcification closely grouped together deep in the right frontal lobe. Each "ball" measured about 1.2 cm. in diameter (Fig. 1). They were interpreted as possibly calcifications in a tuberculoma, a parasitic cyst or calcification in some degenerative condition of the brain. The EEG showed a rather irregular record, with a consistent slow output of high voltage, 5 per sec. waves from the right frontal and anterior temporal regions.

Operation. On Aug. 18, 1952, ventriculography was performed. There was no shift of the ventricular system. The right frontal horn was definitely blunted and shortened. The most posterior one of the calcified masses was adjacent to the body of the right lateral ventricle.

A right frontal osteoplastic craniotomy was immediately performed. The arachnoid membrane and cortex appeared completely normal. A cortical incision was made at about the middle of the 2nd frontal gyrus. At a depth of 2 cm., a thick-walled cavity was encountered, containing yellowish, granular, mucoid material. The calcified masses noted in the x-rays were located around and medial to this cavity. By blunt dissection the cavity and the 5 calcified masses were easily removed from the surrounding brain tissue. A small tear was made in the wall of the right lateral ventricle and this was covered with a piece of gelfoam. Hemostasis was secured, the dura mater was partially closed, and the bone flap was replaced.

FIG. 2. Section of cryptococcic granuloma of right frontal lobe. Many round yeast bodies are seen. Arrow points to budding form. (Kernohan's stain, ×100)

Postoperative Course. The patient received various antibiotics after operation. During the first 9 days, when we were not sure what we were dealing with, streptomycin, penicillin and sulfadiazine were administered. After the diagnosis of cryptococcosis was definitely established, he was given actidione for 3 weeks in doses of 20 mg. intramuscularly, 3 times daily, supplemented by 20 mg. intravenously every other day. Intraspinal injections of 10 mg. actidione were also tried, but were discontinued after 2 injections because signs of meningeal irritation developed. During the last 10 days of actidione therapy he also received 1 cc. of aerosporin every 8 hours, as Carton[2] had reported that the combination of actidione and aerosporin was 8 times as effective as each drug alone. During this period of 3 weeks, the patient received a total of 1530 mg. of actidione and 760 mg. of aerosporin. However, since his temperature and clinical picture had become normal before institution of this combined therapy, we doubt whether the drugs had anything to do with his recovery.

Our suspicions, aroused by the unusual operative findings, led to a culture of the cyst fluid, not only for the usual bacterial organisms, but also for fungus on Sabouraud medium.

The ventricular fluid had a cell count of 1061 RBC and 8 WBC per c.mm.; total protein was 13 mg. per cent. However, the laboratory technician reported "a lot of refractile bodies, which suggested oil globules." An India ink mount of the sediment from the ventricular fluid was immediately done. This showed the presence of spheroid bodies very suggestive of cryptococci or torula. The diagnosis was established when an abundant growth of the organism was recovered from the cyst fluid on Sabouraud medium. The organism was identified as cryptococcus neoformans by the New York State Laboratory in Albany, New York. Paraffin sections of the granuloma and cyst wall also showed the typical organisms, without any inflammatory reaction in the surrounding tissue (Fig. 2).

The patient was discharged 4 weeks after operation. He was recalled at intervals of 3 months for reexamination. Examination of the CSF 1 year after operation showed a cell count of 2 WBC, a total protein content of 28 mg. per cent and normal sugar and chlorides. The centrifuged sediment of the CSF showed no yeast bodies on India ink mounts, and culture of the spinal fluid on Sabouraud medium was negative. Clinically, the patient was entirely asymptomatic and had returned to full time work in a furniture factory.

SUMMARY AND CONCLUSIONS

The history of a patient with a calcified mass in the right frontal lobe is presented. The mass was removed surgically. Histological section proved it to be a cryptococcic granuloma. The organisms were also recovered by culture of the fluid from a large cystic cavity associated with the granuloma, but never from the spinal fluid.

Cryptococcic infection of the central nervous system is usually rapidly fatal. However, there are well-documented cases of patients who have survived for many years. The most recent case, reported by Beeson,[1] concerned a young woman who had a chronic form of cryptococcic meningitis and finally succumbed to it after 16 years. When the infection is in the form of an isolated granuloma without gross involvement of the meninges, we would expect the chances of long-term survival to be augmented by removal of the mass. Our patient is now apparently well 12 months after operation. We certainly do not consider him "cured" at this early date, but because of the unusual features of his case, we feel that this preliminary report is indicated.

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CONGENITAL ROTATION OF THE SPINAL CORD

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A review of the medical literature disclosed no reference to an abnormality of the spinal cord of the type here described.

Case History No. D. 24296. J.G., male, aged 52, was admitted to the Neurosurgical Service on Oct. 27, 1952. Until 3 weeks previously he had been a fit man. On the day his illness began he was lifting a load of wood behind his house when he experienced a tight feeling amounting to a pain at the lower costal margin on each side of the trunk. This lasted for 20 minutes and he continued his work. A few hours later the pain recurred and, in addition, there was a pain in his back in the lower dorsal region. About an hour later he noticed numbness and weakness in both legs of which the right was almost paralyzed; he was unable to walk. The same evening retention of urine developed and he required catheterization. Thereafter his condition remained unchanged until his admission 3 weeks later.

Examination. The abnormal signs were referable to the spinal cord with the exception of a hypospadias at the penis-scrotum junction. (1) The right leg was paralyzed except for a flicker of movement in the hamstrings; the left leg was weak but all movements could be made against resistance. The lower abdominal muscles were weak. (2) The tone in the legs was increased, particularly on the left, and the left plantar response was extensor. (3) On the sensory side there was a complete absence of recognition of painful and thermal stimulation on both sides below and including the 9th thoracic dermatome. Immediately above this was a narrow band encircling the trunk where pin-prick could be recognized but it was not felt normally. Below this level there was an almost complete preservation of vibration and position sense, the only impairment being at the right ankle and foot. The sacral segments did not escape the general sensory involvement.

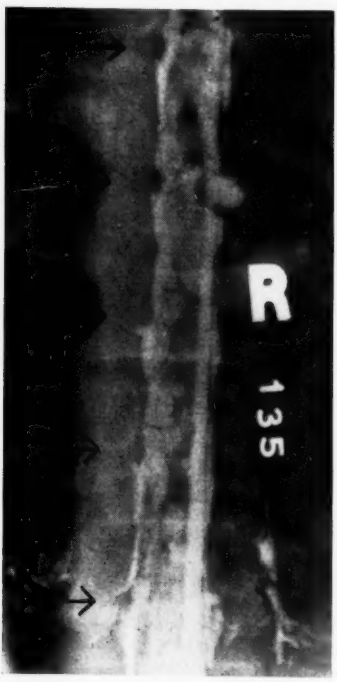

Fig. 1. Myelogram. Compare the appearances here with Fig. 2. Oil can be seen within the large cyst removed at operation, and the arrows indicate the regular indentations of the column caused by the constricting nerve roots.

Investigation. (1) X-rays of the vertebral canal were normal. (2) Lumbar puncture: pressure 180 mm. CSF. Free rise and fall on jugular compression. Proteins 32 and 40 mg./100 ml. (3) Cisternal myelogram showed regular lateral indentations of the column of oil (Fig. 1) and in relation to these filling defects could be seen separate globules of oil of varying size. The significance of this appearance was not grasped before operation. There was no obstruction to the flow of oil up the spinal canal.

Operation. It was decided that exploration of the cord should be carried out, and since there was no myelographic localization the exposure was centred on the cord segments T8–9. The spines and laminae of vertebrae T6, 7, 8 and 9 were removed.

Figs. 2 and 3 illustrate the state of affairs that was discovered. The removal of the extradural fat revealed the spinal dura mater crossed on its dorsal

aspect at regular intervals by cord-like structures of 3 to 4 mm. diameter which could be identified as spinal nerves. Each nerve emerged, covered by its sheath of dura mater, from the left posterolateral aspect of the spinal dura mater and escaped from the vertebral canal through the right-hand intervertebral foramen at the same level. A probe could be passed between the nerve and the dural sac to show that it was a separate structure. Before the nerve escaped through the foramen it expanded into a cystic swelling, the size of which varied in different nerves from an insignificant bleb to a round cyst measuring 8 mm. in diameter. The wall of the largest of these cysts appeared to consist only of arachnoid for it was transparent

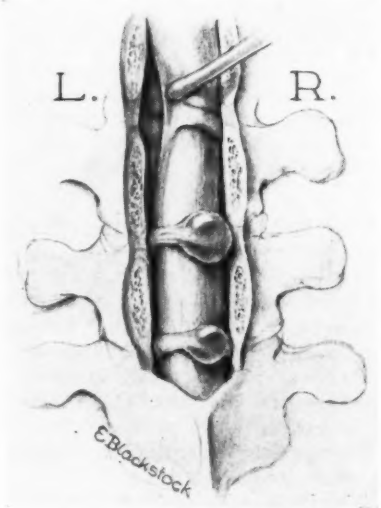

FIG. 2. Appearance at operation (see text). The dura mater has been retracted to show a left nerve root lying on the wall of the vertebral canal.

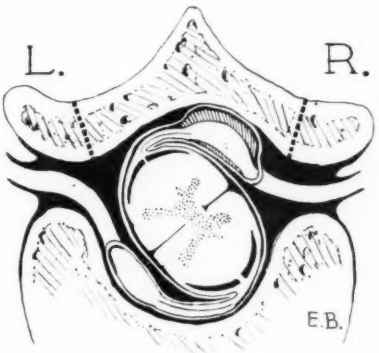

FIG. 3. Diagrammatic cross-section of the anomaly (see text).

and some nerve fibres could be seen glistening through it. The spinal dura mater was pulsating slightly but no pulsation could be made out in the cyst. Retraction of the dura mater towards the right exposed the left lateral and anterior walls of the vertebral canal. Here the intercostal nerves of the left side could be seen emerging from the intervertebral foramina, but each could be traced back into the vertebral canal as it ran at right angles to the long axis of the body closely applied to the wall of the canal and embedded in the extradural tissues until it disappeared beneath the spinal dura mater. By retracting the dura mater on the opposite side (the right) the emergence of these left-sided nerves could just be seen. No cysts were encountered on the left intercostal nerves in the operative field.

The largest cyst was excised between silver clips and the dura mater was opened as far as the nerves above and below would allow. The dorsum of the cord was not revealed, but instead nerve rootlets running diagonally downwards from right to left could be seen. These were the motor rootlets before they had joined the corresponding sensory ones to form the mixed nerve. The ligamentum denticulatum was identified clearly and could be seen attached to the dura mater a little to the left of the mid dorsal line; this was its true right-sided attachment. There was not any gross vascular abnormality. Nothing more was done and the wound was closed.

Progress. Four months after this operation he showed only slight improvement in the movement of the right leg. He was unable to walk and was committed to a wheel chair. The

paraparesis was complicated by flexor spasms. The upper level of sensory impairment had risen to include the 4th thoracic dermatome.

Histology (Professor Eric A. Linell). The section contains a dorsal root ganglion and some dorsal nerve roots. Many of the ganglion cells have an irregularly shrunken cytoplasm in which the Nissl substance stains abnormally deeply. Nuclei are small, pyknotic and frequently eccentric. There is a marked increase in fibrous tissue within the ganglion. One of the nerve roots shows similar severe endoneural fibrosis with loss of myelin sheaths. A second bundle of nerves must have been twisted almost at right angles as part of it has been cut transversely and the remainder longitudinally. Its transverse portion is essentially normal. In the longitudinally cut portion the myelin sheaths are severely swollen and disrupted, their microscopic appearance suggesting operative trauma. The fibrous tissues surrounding both the ganglion and the nerve roots are thickened. Some normal extradural fat is adherent to the outer surface of this fibrous tissue along one edge of the section. A thin, moderately cellular fibrous membrane lies free on the opposite side of the section. This probably represents a portion of the wall of the cyst seen at operation.

DISCUSSION

Rotation of the spinal cord as described in this report has no embryological justification. It is curious that the dura mater and ligamentum denticulatum, both of mesodermal origin, should be involved as well as the ectodermal element, the cord itself. The only other congenital abnormality demonstrated was the hypospadias; radiographic investigation of the chest and abdominal viscera did not disclose further anomalies. It would have been interesting to know at what level the rotation had taken place. The myelograms of the cervical region were completely normal and the shadow of the nerve roots could be seen streaming out equally and at the correct angle from the cord. The lateral indentations formed in the thoracic area by the encircling peripheral nerves are absent from the cervical pictures. It can only be presumed that the rotation took place at the cervical-dorsal junction.

The sudden onset of symptoms may be explained on the basis of inflation of one of the cysts with cerebrospinal fluid at the time of a strain. On the other hand, the cord lesion may have been caused by a spinal vascular accident, although the bilateral girdle sensation of which he complained in the first place does not suggest that this mechanism was responsible. The rise of the level of sensory loss during the weeks succeeding the operation indicates either that a fresh compressive lesion arose in the postoperative period, or that the original exposure, although sited according to the neurological signs, did not coincide with the situation of cord damage. This would not be surprising since the myelogram gave no indication of the appropriate level.

SUMMARY

A case of rotation of the spinal cord and meninges is reported and illustrated. No previous published reference to this anomaly was discovered.

My thanks are extended to Miss E. Blackstock for the illustrations, to Professor E. A. Linell for the histological report, and to Dr. E. H. Botterell for permission to record this case.

CORRESPONDENCE

FROZEN BONE FLAPS FOR GRAFTS

To the Editor:—In a paper entitled "Use of Frozen Cranial Bone Flaps for Autogenous and Homologous Grafts in Cranioplasty and Spinal Interbody Fusion" (*J. Neurosurg.*, 1953, *10:* 380–388), the writer has inexcusably overlooked the important and primary contributions to this subject by H. Elliott and H. J. Scott, "The Bone-Bank in Neurosurgery" (*Brit. J. Surg.*, 1951, *39:* 31–34) and by G. L. Odom, B. Woodhall and F. R. Wrenn, Jr., "The Use of Refrigerated Autogenous Bone Flaps for Cranioplasty" (*J. Neurosurg.*, 1952, *9:* 606–610).

These were called to my attention after this paper was submitted for publication, and the writer offers his apologies to the above pioneers in this field.

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NOTICE

ELECTRONICS AND NUCLEONICS IN MEDICINE

The 6th Annual Conference on Electronics and Nucleonics in Medicine will be held at the Hotel New Yorker, New York City, on November 19–20, 1953. The conference is again being sponsored by the American Institute of Electrical Engineers, the Institute of Radio Engineers and the Instrument Society of America.

A considerable portion of the program will be devoted to diagnostic aids in the fields of blood measurements and brain measurements. One paper will introduce positron emitting isotopes as aids in locating brain tumors. Another will feature three-dimensional x-ray motion pictures as aids in diagnosing heart artefacts.

In addition, one complete session (Friday morning, November 20) will deal with various aspects of blood analysis, including papers on: the exact determination of volume concentration of non-conducting particles in a conducting solvent; an instrument for rapid determination of freezing point depression; blood serum analysis by a quantitative paper electrophoresis apparatus; infra-red CO_2 analysis; and determination of CO action spectra.

A special semi-popular type lecture-symposium Thursday evening, November 19, will consider the field of x-ray cinefluorography and the impact that the new image brightness intensifiers have had on the field. Mr. Sydney Weinberg of the University of Rochester Medical School will conduct this session.

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